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For Contents See Page 1.

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VOL. XXXVI AUGUST, 1926 No. 8

ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

PROGRESSIVE DEAFNESS. THE CAUSATIVE FACTORS AND SPECIFIC DIAGNOSIS.*

DR. DANA W. DRURY, Boston.

While the term "otosclerosis" is generally conceived to indicate a concrete pathological entity, there yet exists a wide divergence of opinion as to the etiology of the condition, and in lesser measure the characteristic symptoms defining it and the proper method of treatment. In regard to the first question, while it may be freely acknowledged that as yet no specific knowledge exists as to the basic exciting cause of the condition, equally much progress has been made in the establishment of the correct prophylaxis of the condition.

It is generally accepted today that it originates in the bony capsule surrounding the labyrinth and that the diverse and at times contradictory findings depend upon the location in the capsule of the primary focus of disturbance. As the condition progresses, however, the several divergent pictures tend to merge and with the fixation of the stapes in the oval window, all cases present much the same clinical entity. While these changes affect the function of hearing, it is nevertheless a chronic nonsuppurative disease. Anatomically, it may be well to remember that the layer in discussion shows only at the promontory, the oval window, the external semicircular canal, and sometimes at the superior canal. The most striking point is the relative inconsistency of the clinical symptoms in contrast to the constant anatomical changes present.

*Read at the Annual Meeting of the N. E. L. and O. Society, Boston, Jan. 28, 1926.

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Many observers have described the changes brought about in the labyrinth by this disease and they all agree that in general, otosclerosis begins by the absorption of bone in some part of the capsule. The region which is supposed to be most commonly affected is that immediately above and in front of the oval window, although any region of the capsule may be affected from the edges of the round window to the walls of the semicircular canals. The pathological process consists essentially in absorption of the old bone followed by the deposition of new-formed spongy bone in its place. The diseased focus is at first sharply marked out from the surrounding healthy bone. The Haversian canals within this area become enlarged, their walls being absorbed by osteoclasts. These changes take place in regular sequence until finally the area cannot be distinguished from the old bone surrounding it. Throughout this entire metamorphosis it is important to observe that there is no sign of an inflammatory action.

Local and constitutional conditions do, I think, play an important part in determining the onset and course of this disease. Therefore, the evidence of heredity plays a very distinct role in the future development of the deafness. Several authors are inclined to regard the hereditary tendency of otosclerosis as of importance in the differential diagnosis against the adhesive processes.

Fundamentally we may say that children inherit, or that the parents transmit, only those peculiarities which are contained as rudiments of the parental sexual cells. The union of the ovum and the sperm cell complete the act of inheritance. The inherited material we may call determinants. These units are hypothetical and ultramicroscopic. According to one theory these aggregations are distributed to different parts of the organism during development and so determine the differentiation of the individual cell. Thus any pair of parents can produce many different combinations, each combination being a child of different characteristics, but nevertheless exhibiting certain broadly general similarities due to environmental influence. It seems reasonable to believe, therefore, that as these gene must be composed of chemical entities they may be transmitted through an individual to the progeny without ever having become active in the intermediate host.

In Bezold's series of cases, he obtained definite information in 52 per cent that one or more members of the same family, sometimes even a whole group of relatives, were hard of hearing. Our series show four in ten cases. Another fact is still more peculiar. This form of hearing failure concerns especially the female sex,

contrary to all other diseases of the ear. According to Bezold's figures again, only 42 per cent of all his ear patients were women, while 57 per cent of all patients suffering from otosclerosis were female. Our records show 75 per cent women and 25 per cent men afflicted.

Believing as we do that otosclerosis is of an hereditary diathesis, we are desirous of establishing a series of presclerotic cases in otosclerotic families, as we are of the opinion that from such a study we will secure definite knowledge as to the etiology, especially in the cases showing an endocrine dysfunction. Further, the endocrine influence is one of indirection. The several units of the endocrine system are among the potent regulators of the general metabolism, hence disturbed function in these as in other and nonendocrine regulators may produce metabolic disturbance. As we believe that otosclerosis is the end result on a constitutional tendency produced by a disturbed metabolism, it follows that we must have cases in which the endocrine element is present and equally those in which it is absent.

Most all observers agree that a perfectly uncomplicated case of otosclerosis, with the bone changes confined to the stapedial region, is easily recognized. In this connection, however, we all meet cases which fall into two other categories, namely, otosclerosis complicated by middle ear catarrh and those cases with foci distributed in parts of the labyrinth removed from the stapes and the annular ligament. Generally, the onset is very gradual and as it advances both ears are generally involved. Frequently the ear in which symptoms appear last is the one in which the progress of the lesion is subsequently most rapid and the deafness most pronounced. This may be observed in cases beginning, or rather first becoming patent during a pregnancy.

As to symptoms, deafness and tinnitus are always complained of at some stage of the disease. Personally, I believe a diagnosis should only be reached through several observations, and not determined on a single examination, no matter how careful this latter may be performed. The ultimate degree of the deafness may be either comparatively slight, or very great, but only in very rare cases does it become so marked that the patient cannot understand a loud conversational voice in close proximity to the ear. When this does happen, I think we may be safe in saying that the primary condition is complicated with degeneration of the auditory nerve through some outside influence.

In advanced stages the hearing tests in our series of cases show the Rinné test negative in over 90 per cent; a prolonged bone con-

duction in about the same proportion, and raised lower limits in nearly 100 per cent, while the lowering of the upper limits is very much less marked. Regarding tinnitus, it was present in 82 per cent, paracusis in 75 per cent, and vertigo in only 30 per cent of all our cases. An endocrine element was established in 68 per cent of the cases examined by the methods described elsewhere. This group can be subdivided into 36.6 per cent for pituitary dysfunction; 16.6 per cent for thyroid disease; and 15 per cent for gonad failures.* We have had no so-called pluriglandular cases.

In conjunction with our findings in the hearing tests, I am desirous of bringing to your attention three other observations, namely, the effect of amplification of sound on the afflicted ear. In other words, nearly all otosclerotics hear music more clearly than the spoken word and with the latter exhibit greater ease over the telephone and through the radio. Secondly, the presence of a reddish blush seen through the normal drum and located on the promontory back of the umbo. When present it is supposed to be indicative of pathological changes in the bone, or in other words, an active osteitis at this point. In our series this observation is nearly a constant finding. Thirdly, and this presentation I have never seen in print, although it may have been observed, is the absolutely characteristic voice production of an otosclerotic. It lacks inflection, is timid in volume, is low in pitch and is apparently devoid of timbre. Many times there seems to be a tendency to aspirate many of the syllables.

In the initial stage of the affection, where the deafness is slight, and where the subjective noises are the prevailing symptoms, we cannot say with certainty whether we are dealing with otosclerosis or with nerve deafness. In such cases, therefore, if we find an hereditary predisposition, we may suspect otosclerosis. The diagnosis is made with more difficulty in those numerous cases in which the membrana is cloudy, atrophic or retracted, and where catheterization shows that the tube is swollen and narrowed to a varying degree. Under such circumstances, the differential diagnosis between otosclerosis and catarrhal adhesive processes of the middle ear often remains indecisive. We may assume, on the other hand, with great certainty that otosclerosis is the anatomical cause of the deafness if we find the membrana tympani only cloudy, the Eustachian tube of normal permeability, the disturbance of hearing progressive and the results of the functional tests characteristic. If

*These percentages are potentially misleading as there were nearly twice as many pituitary as either thyroid or gonad cases in the series. The incidence of the individual endocrine focus at fault is substantially uniform.

the stapes be fixed there will be no difference in the intensity of the sound during compression and relaxation of the air in the external canal (Gelle), whereas if the stapes be free, the sound will be diminished during compression as in a normal ear.

In another connection, I wish to draw your attention to the fact that there are otologists who consider those cases as otosclerosis only which arise in puberty, and are aggravated in pregnancy. Eventually, we will have a series of these cases from which we hope to draw interesting conclusions. Cases have been reported which showed improvement with pregnancy, but these may have been incorrectly diagnosed. We have never seen this type of case.

Politzer was the first to suggest the relation of otosclerosis to the puerperal state. He thought it was a true infective puerperal osteitis. Mayer believed that there was some connection between otosclerosis and pregnancy, as during that time there are other bony outgrowths, the so-called puerperal osteophytes. I think that most of us have observed also, marked dental changes in an otosclerotic pregnant individual. Stein believes that in pregnancy, otosclerosis either may begin or be markedly aggravated. In our cases the first pregnancy seems to be the most vulnerable. Novak, on the other hand, has shown that toxic substances of sex life may affect the sense organs, especially with pregnancy and the menopause. Aschener has shown that an apparent ovarian toxicosis may lead to neuralgias, neuroses and metabolic disturbances. The rapid changes in these cases may be the effect of a toxic neuritis as we know the marked susceptibility of the eighth nerve to any toxic circulating substance.

Stein also emphasizes the influence of sexual abnormalities on the course of otosclerosis. Frey has apparently demonstrated this fact upon circulatory disturbances. In this connection, I would like to recall to your minds the attention being paid to the ear and the generative phase in otosclerotic patients by our colleagues in Europe. In a meeting held in Frankfurt, Stein cited as indications for the interruption of pregnancy: *1.* if both ears are unfavorably influenced at the start of pregnancy; *2.* if there were a decided hereditary diathesis, especially where it has shown in earlier pregnancies, of such conditions as the psychoses, epilepsy, chorea and polyneuritis; *3.* in the event of high grade excitability of the vegetative nervous system, especially cardiac and vasomotor; *4.* in the case of endocrine disturbances, as with struma, latent tetany, pituitary dysfunction, glycosuria and previous menstrual anomalies. The discussion was more or less as follows: Frankel, of Breslau, thought if preg-

nancies could be proven to have an unfavorable effect on the process an interruption of the pregnancy was indicated. He suggested a test pregnancy. Leichter, of Frankfurt, said that the calcium content was often diminished in otosclerosis, and still further in pregnancy and lactation. He suggested general treatment with the ultraviolet ray. Mayer, of Vienna, thought it was important to determine whether the aggravation of hearing loss in otosclerosis was dependent, either on an enlargement of the bony focus in the fenestra ovale, or on a resumption of degeneration of the sound perceiving apparatus, since it is known that otosclerosis involves these two components. Alexander, of Vienna, concluding, remarked that legally it was necessary to have a condition threatening life to perform an abortion. Some time ago, Hammerschlag suggested interruption of pregnancy, especially when the mother is engaged in business and is dependent on her hearing. The latter advises against marriage of even distant relations in otosclerotic families. Burger cites two cases where artificial interruption of pregnancy was advised. The first was that of a female patient who, although pregnancy aggravated her deafness extraordinarily, refused the procedure on ethical and religious grounds; in the second case a renunciation of children was favorable to the hearing of the wife.

CONCLUSIONS.

1. The desirability of making several independent observations of an individual before a positive finding of otosclerosis is made, is emphasized.

2. The consistency of the anatomical findings with the clinical variations of the disease is noted.

3. Otosclerosis shows a large percentage of cases in which an endocrine dysfunction is a causative factor.

4. The value to be derived from a complete study of pre sclerotic cases in otosclerotic families is pointed out.

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416 Marlborough Street.

PROGRESSIVE DEAFNESS. THE GENERAL DIAGNOSIS OF CERTAIN CAUSATIVE FACTORS.*

DR. ALLAN WINTER ROWE, Boston.

The etiological significance of disturbances of the ductless glands in determining the condition known as otosclerosis or progressive deafness, rests upon two basic principles. First, the pathology of the ear must be diagnosed, conforming in the picture it presents to the several generally recognized criteria defining this condition. Second, the diagnosis of aberrant function of an endocrine focus must equally be established by thorough clinical and laboratory studies. The evaluation of the otosclerosis falls primarily in the hands of the skilled otologist and the primary diagnosis of the condition derives from his study of the case and his interpretation of the facts thus elicited. It has remained for us to accept these cases with the diagnosis of progressive deafness already established and then to study them from the standpoint of the demonstration of disturbed endocrine function as a concomitant factor.

The subject-matter of my paper then deals with the establishment of the medical diagnosis of the patient's condition and serves only as a link between Dr. Drury's discussion of certain causative factors in the primary condition and Dr. Lawrence's statement of the results of treatment in those cases which have been demonstrated to exhibit endocrine dysfunction.

Briefly, the method of approach may be outlined as follows: The literature is rich in papers outlining the technic of so-called vital function tests designed to demonstrate disturbed function in some single focus in the body. These tests have been devised, seemingly, as based upon the assumption that a single definite function of the human body finds its genesis and regulation in a single group of cells. Unfortunately, this assumption is basically incorrect and as a result the appearance of a technic of a vital function test with the laboratory and clinical evidence supporting its validity and specificity, is almost always followed by several articles in which the test is shown to be nonspecific, the proof adducing a like array of clinical and laboratory evidence. The sum total of this effort has been, on the one hand, to discredit the individual test, and on the

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other, to destroy interest in further study. True, there are exceptions to the above broad statement, but in the main the specific vital function test has generally failed of its purpose through demonstrated nonspecificity.

From another point of view, however, this inherent nonspecificity offers the soundest possible approach to methods permitting the evaluation of aberrant function. Recognizing that any function, broadly speaking, is in its manifestation no more than the integration of the several influences of a number of relatively unrelated causes, one is able to determine by observation and study the change produced in the function by variation in one of the factors of control, the others remaining normal or constant. The normalcy of the other factors, however, must be demonstrated and not assumed. Further, on the basis of probability, it is highly unlikely that a disturbance in one of a group of factors will influence a whole series of tests by exactly the same quality and quantity as will a similar disturbance in another concomitant focus of control. If criteria of performance can be established by the study of an adequate number of normal individuals using a sufficient diversity of testing methods to secure a relative completeness in the picture, one may then feel that base lines have been established to which can be referred aberrations of function and both directional and quantitative deviations accurately determined. It must be emphasized that no one test alone is adequate to establish a diagnosis except in a few rare instances. The practice, for example, of returning a diagnosis of hypothyroidism on the single observation of a basal rate measurement some 20 per cent below the calculated normal, is wholly misleading. True, a moderate thyroid failure will produce a drop in the basal rate of this magnitude, but equally a functional failure of the ovary, of the anterior lobe of the pituitary, and among nonendocrine entities a partial inanition will produce an equivalent level. The interpretation of each test in terms of a wide variety of other tests offers the only sound approach to the establishment of an endocrine diagnosis. Further, in the general picture the preparation of a complete and accurate medical history, the performance of a painstaking and thorough physical examination, and the compilation of all other clinical observations constitute a basic and fundamental part of the study. It is doubtful if too much information on a case can ever be secured; it is a fact of daily demonstration that too little is frequently obtained. Finally, tests must be quantitative if the maximum of information is to be obtained.

Acting on the general assumption outlined above, very thorough and complete studies involving all of the several essentials already

indicated, were carried out upon groups of normal people so-called sufficiently large to warrant statistical analysis of an elementary sort. These studies established the base lines or criteria of normal performance. In the course of them certain generally accepted procedures were discarded as they were shown to be without meaning in the form in which they usually presented. For example, the several so-called diagnostic blood sugar curves were shown to be conditioned in their shape solely by the relationship of the exciting dose of sugar to the tolerance of the individual. Supertolerance doses, depending upon their degree of excess above the tolerance level, would elicit anything from the normal to the characteristic diabetic curve. Equally, subtolerance doses determined those sugar curves ranging from that of normal shape, so-called, to the straight line in which the test meal apparently exercises no influence on blood sugar level. The characteristic diabetic curve is characteristic only because the test meal of conventional size represents a massive supertolerance dose to the feeble tolerance of the diabetic. In like manner, it was necessary to devise certain tests to permit of correlations not already established in the literature.

With the criteria of normalcy established, the next phase dealt with the study of individuals in whom a concrete and demonstrable pathology had been produced by surgical intervention. Male and female castrates, surgical hypothyroids and pituitaries, were studied carefully and their departures from the established norm, both in amount and direction, carefully evaluated. By these means then it has been possible to produce fairly typical pictures of certain of the more common endocrine disorders. Due weight must be given to the fact, however, that in the consideration of any one so-called endocrine entity there is a wide variety of possible permutations in functional levels from frank hyperactivity to equally established hypofunction and including all of the possible intermediate gradations. Further, such endocrine entities as the pituitary and the adrenal are certainly at least two entirely independent glands, with the pituitary possibly presenting a third. Even with the apparently simple thyroid gland we have secured experimental evidence indicating with reasonable clarity that the hormone regulating basal metabolism is not the only active agent of the thyroid, while the beautiful work of Dr. Abel on the posterior lobe of the pituitary would seem to indicate an agency inherent therein exercising several widely divergent functions. This may be interpreted severally as pointing to the existence possibly of one, more probably of several hormonic agents having a common origin. From this very mutability of the contributing factors and the divergence in func-

tional level of each, it must be patent that no single test can be regarded as more than one interesting part of a highly complex picture, which must be seen in its entirety to establish a diagnosis.

In the light of the foregoing statement, it may not be inapposite to pause for a moment and touch upon the so-called pluriglandular syndrome. In a series of cases now in excess of fifteen hundred which have been carefully studied by these methods, only one, or a vanishing small fractional percent has been returned as a pluriglandular case. The diagnosis in this instance, I feel convinced, rests not upon a duality of primary involvement but upon our ignorance, which today does not permit us to differentiate the primary from the secondary focus. In the widespread disturbances of metabolism intrinsic in the disturbance of any endocrine focus, other members of the endocrine system will naturally participate in the end results arising from destruction of metabolic equilibrium; but to adduce the failing menstrual function induced by thyroid or pituitary failure as an evidence of primary ovarian complicity, would warrant the connotation of an endocrine function for the intestine because constipation is a characteristic of thyroid failure. Returning to the original line of discussion, the third and most recent phase of the work has been the careful study of cases of obscure etiology and the correlation of the laboratory and clinical findings with the pictures produced by disturbed function of known and demonstrated origin. Again, in this connection it is impossible to overstress the necessity of evaluating the various nonendocrine conditions which might equally give rise to the presenting symptom. Only after every possible nonendocrine entity has been eliminated by repeated test and observation have we felt warranted in interpreting the data obtained in terms of endocrine dysfunction. To illustrate again, in many of the pituitary cases a low-grade eosinophilia exists, but such an observation is interpreted in terms of pituitary dysfunction only after protein sensitivity, skin eruption, intestinal parasites, and the several other nonendocrine potential causes have been ruled out by observation and test.

Time is lacking for the presentation of the very numerous factors which go to make up the diagnostic picture. Summarized briefly, it may be said that in addition to the history and physical examination already stressed, careful and complete analyses are made of the urine, including a so-called nitrogen partition determination, as a high residual nitrogen fraction has potential diagnostic significance; careful studies of the blood chemistry and the blood morphology; the respiratory metabolism, urea curve, phthalein test, vital capacity measurements; sugar tolerance, using the rare sugar galactose, and

a large number of other laboratory procedures either applied directly to excreta as indices of metabolic and functional levels, or forming a part of specifically designed function tests. In addition to these procedures, careful examinations in the many highly specialized fields of medicine are carried out as the other observations suggest their possible informative character. Eye, ear, nose and throat, neurological and neuro-otological, X-ray in its many branches of application, surgical, pelvic, psychiatric, psychologic, cystoscopic, cardiographic, audiometric, and a variety of other examinations are made by men specially trained in these several fields. May I be permitted to say at this time that the completeness of the work and the information obtained from it is due in largest measure to the fine co-operation of my several colleagues to whom I take the greatest pleasure in expressing my deep indebtedness and appreciation.

A few words perhaps may be said on some of the more salient points of the differential diagnosis. For example, the thyroid exercises the most profound influence on the basal rate. The pituitary and the ovary run in the same direction but far less magnitude. Taking cases of equivalent severity, ovarian and pituitary failures, would produce basal rates of the order of —20 per cent, failure of the adrenal cortex might lower the rate to —30 per cent or —35 per cent, while the equivalent hypothyroid case might register —40 per cent, or —50 per cent or even slightly lower values. Conversely, the overactive hyperthyroid reaches very high levels above the normal. The hyperpituitary case (anterior lobe) may reach to +35 per cent or +40 per cent. We have never seen hyperfunctional condition of either the adrenal or the gonad, and with the latter gland are frankly skeptical of the existence of a state of overactivity. A libido interpreted to be above the conventional average level is a rather slender foundation upon which to erect a diagnosis of hypergonadism. Turning to the galactose test, we find initially that there is an intrinsic difference in the level of the normal male from that of the normal female, 30 gm. with the former and 40 with the latter being the test meal necessary to excite a transitory but demonstrable melituria. That the female mechanism for the synthesis and storage of this sugar is a factor is too obvious to be denied. The matter is being studied at the present time. With the sugar tolerance we find that the pituitary is the potent regulator of the power of the body to utilize carbohydrate, failing posterior lobe activity raising, overactivity diminishing the tolerance. The thyroid exercises an influence similar in sense but far less in amount upon this function. The majority of thyroid cases in our experience either show a normal sugar tolerance, or at the most one raised

or depressed in quantity far less than in the pituitary cases. The adrenal picture is not entirely clear at the present time as the rarity of these cases and the small number which we have been privileged to see have circumscribed available data. In the majority of cases, however, with apparent failure of the gland there is a drop in the tolerance approximating the order of that observed with the pituitary. With the gonads, on the other hand, we find an anomaly of great differential diagnostic import. Castration or extreme lowered functional activity in the female drops the tolerance threshold to 20 gm., the prepubertal level. With the male, on the other hand, the testicle exercises no influence on the galactose tolerance. It may be said in passing at this point that while the testicle indubitably exercises a most important influence in the years preceding puberty, the only result which we have observed of castration of the adult male is a slight but hardly significant drop in the basal rate and in the blood pressure. The principal influence of this mutilating operation seems to be psychological in character, and from the data obtained from the study of a group of males castrated in the years following maturity, we are wholly unable to offer any significant evidence of the existence of a testicular hormone of any significant potency. The bearing of these findings on popular literature need not be dwelt upon at this time. Other points of significance which can be only touched upon are the influences of endocrine disturbance, on lymphocyte and eosinophile content of the blood, of the differential influence on blood pressure, pulse rate and body temperature, of the pituitary influence on blood uric acid and that of the adrenal on blood sugar, of the thyroid effect on vital capacity, the pseudonephritis of thyroid disturbance, and the usual kidney derangement where the adrenal is involved, and many others of which space forbids mention. Again let me stress the fact that no one of these points is intrinsic in endocrine disturbance alone, and that the nonendocrine entities must be rigorously eliminated before any endocrine interpretation is warranted. To cite a case in point, in a patient recently examined the laboratory and clinical picture were equally indicative of a specific infection or a pituitary dysfunction with the anterior lobe under- and the posterior lobe overactive The earlier biological tests having been inconclusive they were repeated, giving a positive response with both the Wassermann and the Kahn. Later the fact transpired through another source of information that the patient's husband was luetic. In the first report the pituitary possibility was indicated as only worthy of consideration after lues had been ruled out. The second report, based on the additional information, eliminated the endocrine possibility.

Turning to the immediate problem of the evening, we may discuss briefly the influence of endocrine dysfunction on the origin and progress of otosclerosis. As we conceive this latter condition, there must exist primarily a constitutional tendency which, in many instances at least, is of familial origin. This establishes the hearing mechanism in a given individual as a point of lowered resistance, more susceptible to disturbing influences than other portions of the anatomy. The endocrine glands are among the potent regulators of metabolism. They are not unique, they constitute only one group of several. Should there arise an aberrant function in any one of the several important endocrine glands, a logical sequel is the disturbance of many of the delicately adjusted equilibria which are a part of the complex metabolic functions. With such disturbances then the constitutional tendency renders the hearing apparatus susceptible to functional and organic impairment. The correction of the source of the difficulty, namely, the endocrine focus, permits a re-establishment of the normal metabolic equilibria and so removes the exciting cause of the hearing failure. Where no inhibiting anatomical change has taken place, the restoration of much of the lost aural acuity may be expected. At the worst there is an arrest of the process.

Granted the constitutional tendency, other and nonendocrine regulators of metabolism may exhibit disturbed function and produce the same end result. Roughly speaking, we find two out of three otosclerotics exhibiting an endocrine background; the other one is rigorously nonendocrine. As a complement to the foregoing only a small percentage of cases showing endocrine aberration exhibit otosclerosis as a concomitant or resultant defect. In those cases which are endocrine, the establishment of the focus of failure is essential if corrective therapy is to be initiated. Endocrine medication is specific. It is a true replacement therapy. Otosclerotics of thyroid origin, let us say, need not be expected to show improvement on pituitary medication, and equally the exhibition of thyroid extract to the otosclerotic of nonendocrine origin cannot operate vicariously to the patient's benefit and may, as our observations have shown, produce positive damage to the patient's general level of health. The therapy lies outside the range of this discussion. The establishment of the diagnosis by objective and subjective means constitutes the thesis which I am privileged to present to you. It is a first and necessary step in the general consideration of the problem of otosclerosis.

DISTURBANCE OF HEARING DUE TO TRAUMATISM.

DR. J. COLEMAN SCAL, New York City.

Injuries caused by direct or indirect violence to either the cranium or the auditory apparatus may result in deafness.

Injury to the Tympanic Membrane: In direct violence traumatism to the tympanic membrane may result from:

a. A box on the ear which ruptures the drumhead.

b. Unskilled attempts at removal of foreign bodies from the ear canals.

c. Inflations of the ear with compressed air, especially in atrophic or sicatrized drums.

d. In burns caused by dropping of acid, molten lead, or boiling oil into the ear. A deep-seated inflammation results with destruction of aural tissue. This may result in aural impairment with either total or partial occlusion of the external auditory meatus.

e. Penetration of foreign bodies into the ear canals. Injury to the drum or tympanum by projectiles is rare as the curvature of the meatus guards against this. Drury, of Boston, reports in THE LARYNGOSCOPE, May, 1925, the rare fracture of the malleus resulting in impaired hearing. The condition is caused by direct traumatism on the tympanic membrane by the introduction of foreign bodies. The prognosis in these cases is generally favorable.

In indirect violence traumatism to the drum may follow:

a. Sudden explosions. In these cases a considerable amount of gas is liberated suddenly increasing the atmospheric pressure. This ruptures the tympanic membrane by compression.

b. Fractures which pass through the petrous portion of the temporal bone at the base of the skull. These fractures are apt to occur at the natural passages of the skull, transversely through the external and internal auditory meati or longitudinally through the middle ear, depending on the direction of the blow. Fractures of the squamous portion of the temporal bone may injure the drumhead or ossicular chain and open up the middle ear. If infection of the auditory apparatus already exists such fractures can predispose to purulent meningitis, sinus thrombosis, or abscess of the brain (Lewy).

c. A severe blow on the mastoid region may result in marked impairment of hearing. Lennoirs reports thirty-eight cases of mastoid injury in which hearing was totally destroyed in 73 per cent, and partially destroyed in 21 per cent. Acute suppurative mastoiditis has been recorded following fracture of the base of the skull in

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patients who prior to the injury had a chronic suppurative otitis media.

Injury to the Labyrinth: Injury to the labyrinth is seldom direct. It usually follows indirect violence, such as

a. Concussions (explosions and cannon firing).

b. Basal fractures which may extend into the cranial cavity through the mastoid antrum, middle ear, and internal and external meati.

c. Hemorrhage and exudations into the labyrinth, causing pressure upon the oval or round windows.

The symptoms vary from a slight feeling of faintness to a sudden severe pain at the moment of rupture of the drum. This is usually accompanied by a loud tinnitus, followed by nausea, vertigo, and later the hearing is found to be impaired. Bloody or serous discharge from the ear canal denotes fracture at the base of the skull. Continuous oozing of blood occurs in fissure fractures through the bony canal or at the annulus tympanicus. Deafness may appear at once or a few days later, varying from a simple dullness of hearing to a complete deafness with subjective noises and whistlings. Upon examination of the drum in direct traumatism the perforation, which is usually at the lower part of the membrane, appears ragged and irregular. When due to indirect violence the perforation is slit-like or oval and is usually found at the anterior half of the drum. When a hemorrhage occurs into the labyrinth without drum rupture there are symptoms of nausea, vomiting, dizziness, and slight deafness. If the hemorrhage is severe the deafness becomes marked and is usually permanent. This condition may arise in caisson workers and those rising to great heights in balloons.

Prolonged exposure to air conducted sounds of a certain pitch as well as vibrations transmitted from the floor by looms may cause a gradual deafness which becomes progressively worse. This condition, known as professional deafness, is a gradual deterioration of hearing. It develops in riveters, ironworkers and loomweavers. In these cases the symptoms can be accounted for by a disturbance in the circulation of the middle and internal ear, causing a transudation or hemorrhage into the tympanic membrane or labyrinth and sooner or later results in tympanic or labyrinth deafness. Another explanation is the degeneration of the cochlear apparatus involving first the corti organ and then the spiral ganglion and nerves, due to an exhaustive atrophy from overexcitation affecting primarily that part which corresponds to the prevailing sound.

The diagnosis and extent of impaired hearing can be made by the various tests at our disposal. In examining the auditory apparatus

we should take into consideration both the sound conducting and the sound perceiving apparatus. The tests, which will usually gauge the approximate acuteness of hearing, are made by the watch, voice and acoumeter. In order, however, to ascertain the site of the lesion producing the deafness, and to derive information as to the sound producing and sound conducting apparatus, the finer tuning fork tests are necessary.

The Rinné, Weber and Schwabach tests are sufficient for ordinary use for the determination of the perceptions of the upper and lower tone limits. Perception of the lower tone limit is lost in lesions of the sound conducting apparatus, while the upper tone limit is lowered in lesions of the sound perceiving apparatus.

After a careful history, in examining the vestibular apparatus, spontaneous nystagmus, vertigo, fainting, and falling are noted. The induced tests, such as the rotation, pointing, falling, caloric, and electrical, are then made, and in this way the diagnosis of both labyrinthine and intracranial lesions, following middle ear involvement can be made. Complete traumatic deafness with normal static reactions may be central in origin. In these cases Oppenheim believes the central nuclei in the eighth nerve to be the site of the lesion. Kopetzky, however, believes that, on account of the relationship between the two auditory nuclei in the medulla, it is hardly possible that destruction can occur in this locality without injuring other paths and nuclei lying in the immediate vicinity.

The prognosis regarding hearing depends on whether the labyrinth has been involved or not. If suppuration should occur, a permanent impairment of hearing results, accompanied by a chronic otitis media purulenta. If only the drum has been ruptured a slight impairment of hearing may or may not result. When the labyrinth is affected there may be a complete and permanent destruction of the function of the internal ear followed by loss of hearing, vertigo, nausea and disturbance of equilibrium. There is also a disappearance of high pitched sounds. In labyrinth concussion we get symptoms of disturbance of equilibrium, the vestibular test showing a complete loss of excitability.

Severe and even fatal hemorrhage may occur as result of either direct or indirect trauma. Where vertigo follows concussion or fracture of the skull it usually lasts for a long time and is often accompanied by dizziness, headache, deafness, nervousness and various other necrotic disturbances.

In treatment of such cases great harm can be done by injudicious care. Syringing or instilling drops into the ear canals is contraindicated. The ideal treatment is rest in bed and placing sterile cot-

ton in the ear canal. In favorable cases the perforation will heal, and all symptoms will disappear. Even the impaired hearing will return to normal. The patient must be warned of the danger of permitting fluids to enter the auditory canal.

There is practically no way to prevent direct injuries to the ear. Indirect injuries, however, can be lessened if not actually prevented. This can be accomplished by introducing something into the external auditory meatus to stop or lessen the inrush of air. This can be a cotton plug smeared with vaselin, wax or glycerin.

In conclusion, I would add that in many cases of head injuries no other symptoms but vertigo have been complained of. In fact, complete and partial deafness has been known to occur without any evident injury to the vestibular apparatus. Auerbach and M. J. Gottlieb have reported two cases of total deafness due to trauma with normal labyrinth. Where there is a complete loss of function to one labyrinth it may never be noticed, but if the loss of function is incomplete the dizziness is permanent. It also has been observed that tympanic ruptures are more frequent in cases with pre-existing ear conditions, such as scars or atrophic areas. Fewer ear injuries occur in cases with a pre-existing perforation, especially after explosions.

Case 1: B. S., housewife, age 32 years. Previous history negative. On Dec. 1st, she was in an auto which was overturned in a collision. She was removed to the Danbury Hospital (Conn.), where on regaining consciousness she was bleeding profusely from both ears, mouth and nose. The bleeding stopped on the second day, the left ear becoming dry, but the right ear commenced to discharge a bloody tinged fluid which later developed a foul odor. When I first examined this case on Dec. 8, 1925, I found a facial palsy of the right side with a large hematoma over the right malar region. There was also a slight swelling and tenderness over the right mastoid region. The nose was negative. The throat showed the right faucial pillar not as freely movable as the left. The tonsils were cryptic and diseased. The tongue appeared slightly pushed towards the right. The eyes showed the pupils to be equal and reacting to light and accommodation. The fundi were clear except for a slight congestion of the retinal vessels. There was no nystagmus or occular ptosis. There was no sensory or motor disturbance. There was no adiachokinesis. The reflexes were equal and active, no kernig present. She complained of severe headache, dizziness and diminished hearing on the right side.

Otological Examination showed the left ear dry with a blood clot in the auditory canal. The drum was obscured by blood crusts,

which, when removed, showed a healed perforation at the lower qaudrant. Hearing was in no way disturbed. The right ear showed a profuse, thick, foul smelling discharge which when cleaned revealed a large perforation which had destroyed almost one-third of the drum. The rest of the drum was flat, and there was no sagging of the upper roof canal. Hearing tests showed a considerable diminution in air conduction. Caloric and rotation tests elicited a normal labyrinth response.

A diagnosis of a fracture of the petrous portion of the temporal bone was made, with involvement of the seventh nerve. The other symtoms of mastoid tenderness, purulent foul smelling aural discharge, percussion tenderness over the right side of the skull and facial involvement gave a picture of a suspicious subdural or epidural abscess and on Dec. 16 she was admitted to the Mt. Sinai Hospital for observation. She remained at the hospital three weeks, during which time her symptoms began to clear up. The foul smelling aural discharge ceased. The facial paralysis gradually disappeared and except for headache she felt well.

Wassermann and urinary tests were negative. X-rays of the skull were negative. A lumbar puncture removed 8 c.c. of clear fluid under normal pressure. The spinal fluid showed a cytology of 6 cells with negative globulin. The temperature varied from 99 to 100 while the respiration was from 18 to 24. The mastoid tenderness completely disappeared and she was discharged from the hospital Feb. 6.

In this case all the symptoms definitely pointed to fracture of the petrous portion of the temporal bone involving the seventh nerve. The labyrinth was not involved. The middle ear was involved, due to the exposure to infection by the ruptured drum. This middle ear infection caused considerable loss of hearing by air conduction.

Case 2: O. T., school girl, age 17 years. Previous history negative. On July 4, 1925, she was struck by an auto and knocked unconscious. She awoke at the White Plains Hospital after being unconscious about twenty minutes, and found that she was bleeding profusely from the right ear and mouth. The bleeding stopped the next morning and was not followed by any purulent discharge. She was discharged from the hospital on the fourth day, at which time she noticed she could not hear well with the right ear, and that she was quite dizzy.

She came to my attention on Sept. 1, 1925, at which time the complaint was only of difficulty in hearing with the right ear.

Otological Examination: Right ear showed a scar at the anterior quadrant of the drum near the floor of the canal, result of a per-

foration which had healed without infection. The drumhead appeared to be changed in both color and consistency, looking thick and not as pearly as it should be. The light reflex was not disturbed. The rotation and caloric tests showed a normal functioning labyrinth. The hearing tests showed a definite amount of diminished hearing of the right ear while bone conduction was prolonged, thereby showing involvement of the middle ear.

In this case the labyrinth was not involved. The disturbed hearing is due to the hemorrhage into the middle ear, rupturing the drum. The prognosis is apparently good, although hearing will never return to normal.

Case 3: A. T., school boy, age 8 years. Previous history negative. On July 21, 1925, he was struck by an auto and knocked unconscious. He was removed to the Gouverneur Hospital, where, several hours later, he regained consciousness. Examination at this time disclosed a marked hemorrhage from the nose and the left ear. A spinal tap was made and clear fluid obtained, the cell count of which was 14. X-rays and Wassermann test were negative. The bleeding from the left ear stopped on the second day, the subsequent discharge being recognized as cerebrospinal fluid. This stopped on Aug. 18, when it was replaced by a foul smelling purulent discharge.

A diagnosis of fracture of the base of the skull behind the petrous portion of the temporal bone was made.

Ophthalmic examination, Aug. 31, disclosed a paralytic squint of the external rectus muscle. The eyegrounds showed a slight edema of the disc at the right side and a slight atrophy of the left disc.

Neurological examination on Aug. 4, 1915, showed the left pupil larger than the right, both reacting normally to light and accommodation, with a weakness of the external rectus muscle present. No abnormal reflexes or involuntary movements present. This boy appears to be congenitally deficient, the forehead being very narrow. The mouth is kept wide open and his intelligence is below normal.

Otological Examination: Right ear normal. Left ear showed a scant foul smelling discharge. The drum showed a perforation the size of one-third the drum and situated anteriorly. A marked unilateral deafness was noted. Sound perception by bone conduction was considerably diminished, while the caloric tests showed a hypoexcitability of the semicircular canals. These tests confirmed the diagnosis of labyrinth involvement. Treatment consisted in rest in bed and irrigation of the left ear. The symptoms cleared up except for the aural discharge.

Case 4: J. D., school boy, age 11 years. Previous history negative. On Aug. 15, 1925, he fell from a first story window and was

removed to the Gouverneur Hospital in a semiconscious condition, from which he could easily be aroused and was able to answer questions. Examination at the hospital showed a profuse bleeding from the nose and left ear, together with a bilateral nystagmus. The bleeding from the left ear stopped on the second day, when clear fluid, which upon examination proved to be cerebrospinal fluid, appeared. A spinal tap, taken Aug. 17, resulted in clear fluid obtained under 20 m.m. mercury pressure with a cell unit of 14. Subsequent spinal taps, taken Aug. 18, 19 and 20, obtained clear fluid under 8 m.m. mercury pressure, with a count of 14 cells. X-rays taken Aug. 17 were negative. Wassermann test was negative.

A diagnosis of fracture of the skull with left aural involvement was made.

Otological Examination: Right ear was normal. The left ear showed a scant clear, watery discharge with the external auditory canal filled with small dried particles of blood and macerated epithelial cells. The drum showed a large oval perforation at the lower anterior quadrant, the margins of which were well defined and slightly inflamed. Hearing was slightly diminished, bone conduction being increased. The labyrinth was unaffected, as shown by the normal vestibular reactions. The perforation healed completely without any aural discharge. The child's hearing was practically normal on the discharge from the hospital.

Case 5: D. B., salesman, age 27 years. On July 2, 1925, while driving in Asbury Park a truck collided with his car and he was thrown 40 feet, striking against a wall. He was taken to the Memorial Hospital (Long Branch), where he remained intermittently unconscious for two days. When he awoke he found he was still bleeding from the left ear.

Otological Examination (Sept. 5): Left ear showed a large scar at the center of the drum, with an apparent fixation of the drumhead. The hearing tests showed increased bone conduction, while low pitched sounds could not be heard. The watch could be heard only on contact. The voice test was heard at 2 meters. There was no vertigo or dizziness. The vestibular tests were normal except for a quick excitability. A diagnosis of fracture of the base of the skull without any labyrinth involvement was made. The amount of diminished hearing in this case is undoubtedly permanent because the formation of adhesions in the middle ear cause a fixation of the drumhead.

44 W. Seventy-fourth Street.

CLINICAL OBSERVATIONS ON THE USE OF THE AUDIOMETER IN TESTING THE HEARING.

DR. WILLIAM NEMZEK and DR. HAROLD HAYS, New York.

We have several methods of testing the hearing; namely, the tuning forks, watch, spoken voice and whisper. These tests are all valuable to the individual making the test, but as a whole they are of less worth when we try to designate our findings in a graphic or written way to pass on to another confrere.

In a city it is impossible to find the quiet, large rooms necessary to make the voice test, and if we did have ideal conditions, these tests could not be checked by another otologist, because the personal equation counts for so much.

One physician can rarely correlate his finding with that of another when the usual methods are used. We are fortunate, therefore, to have at present an instrument, the audiometer, which in our practice seems to give us much more exact information than any other test. We use the Western Electric 2-A Audiometer. Standardized charts are supplied with the instrument on which we can record the patient's acuity of hearing for the various tones of the musical scale. These can be scanned in a few moments and show us a definite picture of the amount of hearing lost or retained, meaning as much also to the physician in other cities as to the man who has made the tests.

The greatest advantage of the audiometer is the absolute quantitative analysis it gives us, which is so important in noting whether our treatment is beneficial or detrimental. By a simple test we can tell if a gain in hearing is psychological or real. This is very important as we often see patients who think they have been benefited by unscrupulous practitioners of various kinds; whereas they have not been helped in the least. It also helps us very much in ascertaining when we shall stop treatment in cases of progressive deafness, as overtreatment in these cases is worse than no treatment, because it often hastens the process.

The audiometer has one drawback. In children up to 6 or 7 years it is not accurate and is more tedious in testing the hearing than other methods, because it serves as a toy and it is impossible to have them signal when the tones are no longer heard. It is, however, a great help in testing the hearing of children who are so-called deaf mutes. In our hands the whispered and spoken voice are better methods of

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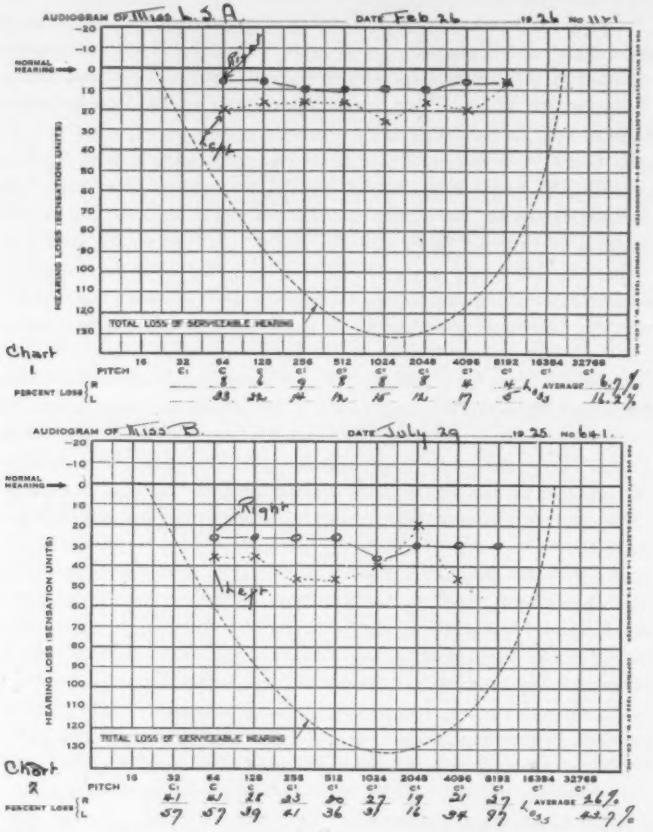
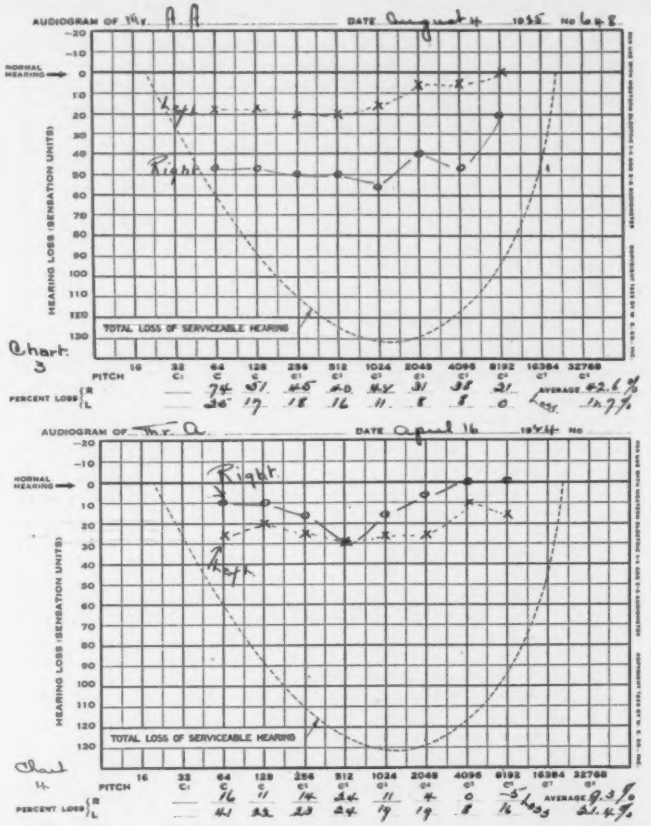
testing the hearing of children who are not fit subjects for the audiometer.

The various audiometers have a signal which is regulated by the patient telling us when they hear and when the tone has entirely



died away. We find this rather disconcerting to some patients and requires much time in rechecking, as the patients forget to press or release the button at the proper time; hence the test is of no value and in order to have it accurate requires many retrials. We simply have the patient say "yes" when he hears and "no" when the last sound is heard. This does not require the patient to think of listening and doing something mechanical; thus he can give his undivided attention to the sound he hears and its disappearance.

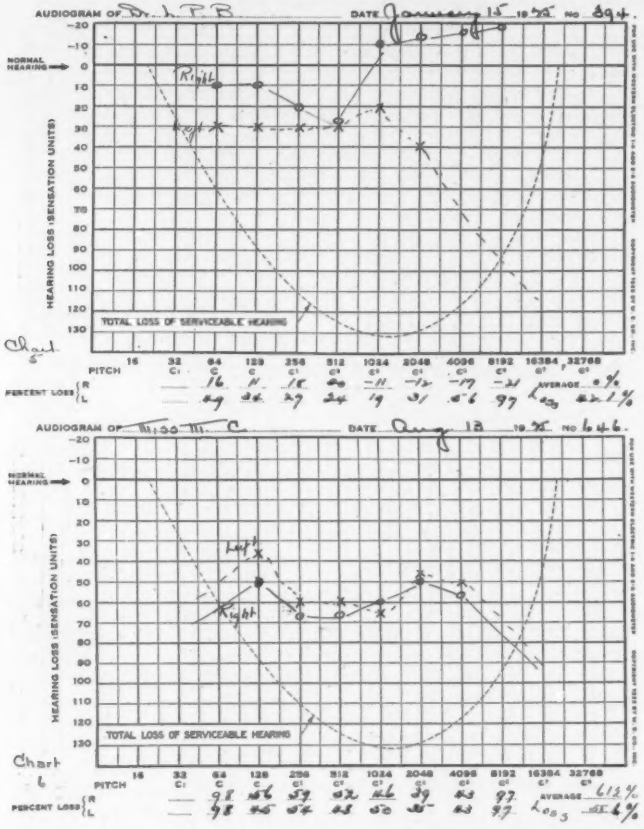
The audiometer test is not always accurate in patients having head noises, as very often they think they hear the instrumental tones when in reality it is head noises which they are hearing. This error can be corrected almost entirely by means of a button which shuts off the instrumental tones when pressed upon, returning when the button is released.


AUDIOGRAM OF Mr. A. A
DATE August 14 1925 No 648
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
Left
Right
TOTAL LOSS OF SERVICEABLE HEARING
Chart 3
PITCH
16 32 64 128 256 512 1024 2048 4096 8192 16384 32768
C C1 c c1 c2 c3 c4 c5 c6 c7
PERCENT LOSS
R 74 51 46 40 44 31 38 21 AVERAGE 42.6 %
L 36 17 18 16 11 8 8 0 Loss 14.7 %
AUDIOGRAM OF Mr. A.
DATE April 16 1924 No
Right
Left
TOTAL LOSS OF SERVICEABLE HEARING
Chart 14
PITCH
16 32 64 128 256 512 1024 2048 4096 8192 16384 32768
C C1 c c1 c2 c3 c4 c5 c6 c7
PERCENT LOSS
R 76 11 14 24 11 4 0 -5 AVERAGE 9.3 %
L 41 22 23 24 19 19 8 14 Loss 21.4 %

In this paper we will show a few of the graphic pictures representing the acuity of hearing in various types of pathological conditions of the ear. Chart No. 1 represents the impairment of hearing in a case of impacted cerumen. Such patients frequently complain of very serious loss of hearing. The chart shows a moderate diminution in hearing for the lower notes (indicating a defect in

the conduction apparatus), but a gradual rise to normal for the higher notes. When the cerumen is removed, in the majority of cases, the hearing returns to normal, particularly if a mild massage has been given to the drum.

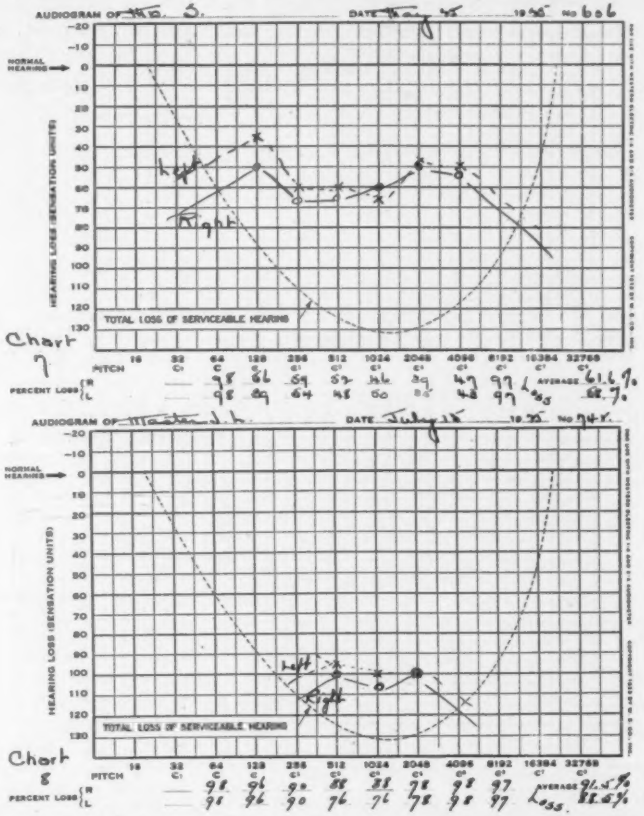
Chart No. 2 is the picture of an acute purulent otitis media in the left ear. We find that in most cases, along with the loss of



hearing for the lower notes, we usually have loss of hearing for the higher notes and in most cases complete loss after fork 4096-C5. We attribute this phenomenon to congestion of the inner ear concurrent with the middle ear inflammation. The higher notes often return to normal quite rapidly after proper drainage is instituted. The right ear in this case also shows a loss of hearing. Examina-

tion showed a tubotympanic congestion, which cleared up in less than forty-eight hours.

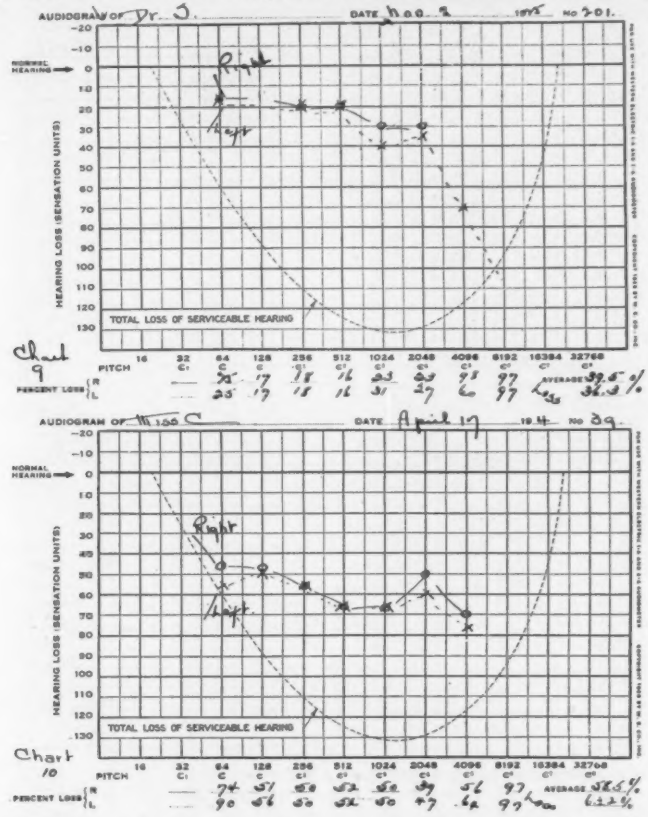
Chart No. 3 was taken from a patient operated upon for acute suppurative mastoiditis (right). The ear had been discharging two weeks previous to operation. One should note the marked loss of hearing for the lower notes and how the hearing for the higher notes almost reaches normal at fork 8192-C6. The greater loss of



hearing for the lower notes in this case over the case of acute otitis media is due to the greater amount of pathology present in the conductive apparatus. In spite of a marked suppurative mastoiditis the higher notes are near normal. When one compares the curve on this chart with the curve on Chart No. 2, one can readily understand the importance of early free drainage in acute middle ear

disease. It, most assuredly, in many cases, is complicated by a congestion of the inner ear.

As we are now speaking of labyrinthine congestion concurrent with middle ear inflammatory diseases, we present Chart No. 4, which is a bilateral, acute tubal inflammation without tympanic congestion, and Chart No. 5 represents a tubal inflammation of the left

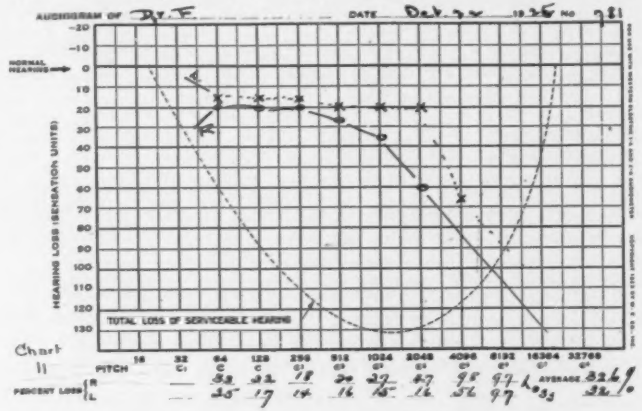


ear which progressed to a tubal tympanic congestion with an occasional dizzy spell. One should note the marked drop of the higher tones from zero line. The tympanic congestion in this case subsided after one treatment and incision was not necessary. The inflammation in the tube persisted several days.

We now come to those cases in which we find the audiometer of the most value; namely, chronic progressive deafness due to chronic

hyperplastic and chronic hypertrophic otitis media. These cases present various graphic pictures. Some progress in a year or two to as great an impairment in hearing as others do in ten or fifteen years. This is not the rule, however.

Chart No. 6 represents a case of chronic progressive deafness of three years' duration. One will see that the higher notes are not involved. In other words, there is interference with sound conduction. Chart No. 7 is taken from a patient who first noticed deafness fifteen years ago. The high notes are also markedly diminished. We find that in most cases of progressive deafness the high notes show a loss after 2048-C4 and in many cases we find a downward trend from 64 to complete loss at 2048-C4.



We treat a great many cases of progressive deafness and the audiometer curves serve to show us just how beneficial certain lines of procedure are and also tell us when the case is being overtreated or mistreated. It is most interesting to see a gain for certain notes; but the most gratifying thing, in the very advanced cases, is to find on retesting the hearing after a course of treatment, that the patient is able to hear a note he did not hear before treatment. This is very important, for it shows us that we are benefiting the patient and further treatment is justifiable.

In so-called deaf mutes, who have residual hearing, the audiometer is indispensable. We can tell by the expression on the face of the patient if he has the slightest trace of hearing. It is most important for us to ascertain if such unfortunates have a trace of hearing, for if we can establish such a fact, we are able to manage such patients along the lines of auditory re-education. The speech

area lies between 236-C1 and 2048-C4 and it is in this area that one may find "islands of hearing" to work upon.

Dr. Max A. Goldstein, of St. Louis, has demonstrated what he can do with such cases and the results are indeed worth the effort. Chart No. 8 represents a deaf mute with retention of some hearing. It would be sad to consign such a child to the sign language group. On examination it may be possible that the child only felt the vibrations of the audiometer. However, he gave no signs of feeling or hearing with the maximum vibrations of the other notes; consequently we feel the notes were heard.

Our experience proves that in otosclerosis we must correlate the graphic chart with the history and clinical findings. Chart No. 9 is representative of an early case of otosclerosis diagnosed as such by at least a dozen good men in New York City. The chart shows loss of hearing of moderate degree of the lower notes, with a sudden loss of hearing, starting at 2048. This is almost the invariable rule in this disease. In some cases, the loss is greater; in others, less. It is interesting in this case that our examination showed a small bony protuberance from each superior wall of the bony canal adjacent to the drum membrane. It is evident the process involves the inner ear, as the higher notes are lost. Chart No. 10 is one of advanced otosclerosis with greater loss of the higher notes.

Many of our patients complain of missing some phone call, high pitched musical sounds or singing only and wish to have their hearing tested. Such patients are usually over 40 years of age. Chart No. 11 represents this type of case. The lower notes are quite normal, while the higher ones are lost abruptly after fork 2048-C4 or 4096-C6. The patient hears the ordinary spoken voice as well as any normal person. We consider such cases as due to an absorption of toxins into the system, causing a reaction on the auditory nerve as well as acting as a depressent to the entire nervous system.

CONCLUSIONS.

1. The audiometer is an instrument which gives us a rapid, accurate method for testing the hearing.

2. It shows in what part of the hearing apparatus the pathological lesion is present.

3. It indicates when conductive and perceptive lesions are combined.

4. Its greatest value lies in its ability to definitely tell us whether the individual under treatment is receiving any benefit from treatment.

5. It allows of comparisons of hearing tests by different individuals.

22 West 74th Street.

FAMILIAL EPISTAXIS.

Dr. Louis Blumenfeld, Brooklyn.

Familial epistaxis is a disease rarely met with. In carefully looking through the literature on this subject I failed to find the report of any individual case. Osler in his textbook makes mention of the disease in the following manner: "There is a family form (of epistaxis) in which many members in several generations are affected. I have described a remarkable hereditary multiple telangiectasis, a special feature of which is recurring epistaxis. This disease has nothing to do with hemophilia, with which it has been confounded. The bleeding occurs from the telangiectasis in the nasal mucosa, and from those in the lips, tongue and skin. A severe anemia may be caused by the loss of blood." The rarity of the disease, the severity of the symptoms produced and the ease with which the bleeding was permanently stopped prompted me to report this case. The patient bled from no other part of the body except the mucosa of the nasal septum.

On Dec. 6, 1925, this patient, a Russian, age 45 years, and by occupation a mattress maker, came to my office with a facies of pernicious anemia and very dyspneic. My first impression of him as he entered my office was either a severe secondary anemia or one of the primary anemias. He was extremely pale, almost cadaver-like. The skin of his face appeared to be edematous. While he was removing his overcoat his nose began to bleed. His wife, who was with him, said: "That is why we are here." I immediately packed his nose with an adrenalin tampon and ordered him to rest for a while. After resting for about half an hour I removed the nasal packing. No bleeding followed. I examined his nose carefully. The mucosa of both sides of the septum was very pale. There was a spur on the right septal wall at the junction of the septum and nasal floor. Otherwise no abnormalities were noted. No dilated blood vessels were visible. Those portions of the mucous membrane on the septum not adrenalized seem to bleed easily to the touch of the probe.

On account of his marked weakness and severe secondary anemia I immediately ordered him to the hospital. For some reason or other he did not reach the hospital until Dec. 9. During this interval he

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had daily nosebleeds, which he had learned to control by packing his nostrils with absorbent cotton saturated with adrenalin.

He was admitted to the Jewish Hospital on Dec. 9 at about 3:30 p. m. I will outline his complete history as noted by the house surgeon.

Chief Complaint: Epistaxis, weakness, dyspnea, cardiac palpitation.

Family History: Mother died at age 67 years, due to some renal involvement. Father is alive at 80 years. He has three brothers and three sisters. All are living. All, with the exception of one brother and one sister, suffer from epistaxis. His wife is alive and well. She has had six induced abortions, one of which resulted in postabortal sepsis. As a result of this sepsis a blood transfusion was required, for which he (the patient) was the donor. Almost everyone in the family on his mother's side suffer from nosebleeds. He has four children; all are sufferers of recurrent epistaxis.

Personal History: Patient has had measles and pneumonia. Had a sudden attack of dizziness and numbness of the extremities seventeen years ago. Has had no recurrence. He has had frequent attacks of cardiac palpitation during the past eleven years. His gastrointestinal system is negative. He has had a chronic cough for fifteen years. His cough usually starts in the spring and autumn, lasting about six or seven weeks. It is productive of a small amount of grayish sputum. There was never any hemoptysis. He has nocturia and polyuria. He has difficulty in starting the stream, but without pain. There never was any hematuria. He is a poor sleeper and has occasional headaches. He lost ten pounds in the last six weeks. He had a Neisser infection at the age of 19. He denies lues.

Present Condition: The patient has had nosebleeds as long as he can remember. For the past fifteen years they have occurred more frequently; sometimes two or three times a day. He never bleeds anywhere else. He does not bruise easily. For the past six weeks the patient worked very hard and his nosebleeds have been very severe. Five weeks ago he had one that lasted for three-quarters of an hour. Because of this loss of blood he has become progressively weaker. He gave up work four days before admission to the hospital. With this weakness dyspnea has appeared. Whenever the patient has a severe nosebleed he becomes dyspneic. For the last six weeks he has had palpitation almost constantly. When he gets a severe nosebleed he gets a feeling of tightness in his extremities.

Physical Examination: A moderately obese, male adult, very pale. No dyspnea, no cyanosis. Pupils are equal and react to light and accommodation. There is no ocular paralysis and no nystagmus. The heart sounds are distant, pulse 80, no murmurs. The skin and mucous membranes are extremely pale. There are no purpuric spots anywhere. Examination is otherwise negative.

Laboratory Analyses: Urine: negative. Feces: negative for blood, ova or parasites. Wassermann: negative. Blood count and examination: Red blood cells, 2,760,00; hemoglobin, 44 per cent; color index, 0.8; platelets, 104,000; white blood cells, 9,500; bleeding time, 2 minutes; coagulation time, 4 minutes; fragility, hemolysis begins at 0.42 salin; hemolysis complete at 0.34 salin. Differential: polymorphonuclear neutrophyles, 58 per cent; eosinophyles, 1 per cent; basophyles, 2 per cent; small lymphocytes, 19 per cent; mononuclears, 8 per cent; transitionals, 11 per cent; hybrid cell, 1. Bilirubin (blood specimen): direct, negative; indirect, 1 unit (1-200,000). For blood transfusion: type four.

During the first three days in the hospital he had daily nosebleeds moderate in character and easily controlled by a nasal pack saturated with adrenalin. These days were spent in studying the case clinically and waiting for the completion of the laboratory reports. He had no temperature at any time.

Dec. 13, 1:45 a. m., patient had a profuse nasal hemorrhage, during which he lost 16 ounces of blood. It was finally controlled by a nasal tampon. He bled from both sides of his nose. Patient very pale, covered with cold perspiration. Morphin sulphate gr. ⅙ given by hypo. The pulse was 64 and weak. Hot blankets applied.

Dec. 13: Seen in consultation with Dr. Louria. Medical opinion: patients presents no distinct clinical entity beyond the picture of extensive hemorrhage. The blood picture is in no sense specific, presenting only a moderate degree of thrombocytopenia. Suggestion: Blood transfusion, calcium chlorid medication and local attention.

Dec. 13: 10 c.c. of afenil given intravenously.

Dec. 14: Transfused with 500 c.c. blood by direct method. After the transfusion the general condition appeared better. Had a slight nosebleed from the left nostril, which was easily controlled by a tampon.

Dec. 15: Slight bleeding from the right nostril, controlled by cauterizing with silver nitrate caustic. General condition somewhat better.

Dec. 18: Moderate bleeding from both sides of the septum. Both nostrils packed and patient taken to the operating room. Under local anesthesia both sides of the nasal septum were extensively cauterized with the electric cautery.

Dec. 18 to 21: No bleeding, patient's condition much better.

Dec. 22: Slight blood-tinged serous oozing probably as a result of separation of the slough and crusts. Patient discharged from the hospital.

Dec. 24: Patient came to my office. He informed me that he had not bled since he left the hospital and that he felt better and stronger. His color was better.

Dec. 26: He came to my office and stated that during these two days he had lost about five drops of blood each day while attempting to clear his nose of crusts.

Dec. 28: He lost several drops of blood daily from separation of crusts. On removing several annoying crusts from the left side of his nasal septum, slight bleeding occurred. I cauterized this area with the electric cautery.

Jan. 1: On removing several crusts from the right side of the septum, slight bleeding occurred. Here again I used the electric cautery.

Jan. 5: No bleeding from the nose since his last visit. He was discharged. He said he intends going to Lakewood for a two weeks' rest and then return to work.

He has not bled since Jan. 1.

I believe this case is of sufficient interest to merit the attention of the profession for the following reasons:

1. It is a rare type of epistaxis.

2. The unusually wide familial distribution (as noted in the history).

3. The long-lasting character of the recurrent nosebleeds (since childhood).

4. The ease with which the epistaxis was controlled.

5. The severe secondary anemia produced by this condition.

1564 St. Marks Avenue.

A NEW METHOD OF ANESTHESIA FOR OPENING PERITONSILLAR ABSCESS.

Dr. Gordon D. Hoople, Boston.

Surgery for the relief of peritonsillar abscess is usually done under local anesthesia for reasons which are obvious. Application of a local anesthetic in the presence of an acute peritonsillar abscess is a painful procedure. It often adds to the discomfort of the already pain-weary patient. The application of cocain to the inflamed, mucus-covered surface is often ineffectual owing to the pathological condition present. The injection of novocain or similar substances is often as painful as the introduction of the knife itself. In addition to these difficulties the jaw must be held or pried open for each application of the anesthetic. Even when these methods are carried out it is somewhat frequent to see patients writhe with pain when the knife is finally used.

Because of several such personal experiences, the writer began looking about for a better method. While working on the anatomy of the nose and throat a few months ago, the distribution of some of the branches from the sphenopalatine ganglion suggested the possibility that cocainization of the ganglion might help in solving the problem.

The anatomy and relations of the sphenopalatine ganglion, other than those concerned with the soft palate and tonsil, are not within the scope of this paper. However, three of its branches of distribution have some of their terminal fibres in the tonsil and in the soft palate adjacent to the tonsil. They are the palatine nerves; the anterior, the middle and the posterior.

"The large anterior palatine nerve emerges on the undersurface of the palate through the greater palatine foramen, and at once separates into numerous branches for the supply of the mucous membrane of the soft and the hard palate.

"The middle palatine nerve descends through a small palatine canal, and, piercing the pyramidal process of the palate bone, is distributed to the mucous membrane of the soft palate, uvula and palatine tonsil.

"The posterior palatine nerve consists of one or more small twigs which pass through lesser palatine canals, and supply branches to

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the mucous membrane of the tonsil, soft palate and uvula." (Cunningham.)

It will be seen that these nerves supply the upper part of the tonsil and the soft palate adjacent to it. Knowing that these nerves all originate from the sphenopalatine ganglion, it seemed that cocainization of the ganglion of one side should diminish sensation in the soft palate of the same side and that this would aid in securing a better anesthetization in opening a peritonsillar abscess.

With this in mind a short series of ten cases was tried with the use of this method. Although there was some sensation still present at incision, the findings in each case seem to justify the procedure. The ganglion was cocainized in the usual manner, no special technique being used. A 10 or 20 per cent solution of cocain was applied on a pledget of cotton in the region of the ganglion and left there at least ten minutes. After four or five minutes at least five of the cases volunteered the information that "it doesn't hurt so much to swallow now." The others agreed this was so upon questioning. After cocainization, the abscess was opened with a bistoury and drainage obtained by opening the wound with forceps.

It may be argued that the ten cases were all stoics and would have withstood well the pain of any other method. There may be some truth in this but the cases were picked at random and some were rather neurotic individuals. There is no way of proving this point in the majority of this group, for no one of them had previously suffered with a peritonsillar abscess. However, two of the number had been incised before they came to the writer. In both, no pus had been obtained according to the patient's story.

One of these was the last of the series. Mr. J., age 40 years, came to the emergency ward of the Massachusetts General Hospital, stating that he had suffered with a sore throat on the left side for four days; that he had not slept for two nights and had been able to drink nothing for the past twenty-four hours. The day before entrance he had had his throat "cut three times" without relief. When asked if the cutting caused pain, he replied that it did hurt—very much.

He was evidently suffering considerably. He was an obese subject, weighing close to 300 pounds, though only of average height. He had a short, thick neck, which made examination difficult. It was not easy to get the mouth open wide enough to view the faucial region. It was seen, though, that a peritonsillar swelling was present on the left side which pushed the uvula to the right so much that the uvula was crowded into the right tonsil, thereby cutting off

the view of the posterior pharyngeal wall. With the history, examination and the fact that he had a temperature of 103.6,° a diagnosis of peritonsillar abscess left was made. Further manipulation in the mouth was left till after the cocainization of the sphenopalatine ganglion on the left. The ganglion was cocainized in the usual manner with the use of a cotton plug moistened with 10 per cent cocain.

After the plug had been inserted five minutes, the patient voluntarily remarked, "I could drink a glass of water if I had one. It doesn't hurt so much to swallow."

Following another five minute interval on inserting a tongue depressor the mouth could be opened so that a satisfactory view could be obtained without discomfort to the patient. The abscess was opened with a bistoury and the edges separated with forceps to allow for free drainage. The patient said that he felt the incision, but it did not hurt as did the previous attempts. He made an uneventful recovery.

SUMMARY.

It is not claimed that this is a painless process for incision of peritonsillar abscess because some sensation is still present at incision, but it is so much less painful than most other procedures and so much easier to carry out, that it seems to be the method of choice. Its advantages are:

1. After the diagnosis is made the patient does not have to open the mouth till the operator is ready to incise.

2. A better view can be obtained because the patient can open the mouth wider without pain.

3. In the hands of the writer it is much less painful than the other methods of local anesthesia.

The writer wishes to express his thanks to Dr. H. P. Mosher in making it possible for the writer to see these patients and for his sympathetic co-operation.

243 Charles Street.

SUPERIOR CERVICAL SYMPATHETIC GANGLION SYNDROME.

Dr. F. E. Palmer, Sterling, Colo.

During the past two years I have observed several cases presenting a syndrome which has not before been described. All of these cases except two gave a history of a past or present nasopharyngitis, in some more pronounced than in others. In the two which failed to give a positive history, inspection showed the presence of a low grade of inflammation in the nasopharynx. Three of these cases were referred to me for a mastoid operation because of the earache and unilateral headache, and one had already had a myringotomy done. All of the cases cleared up and only one, to my knowledge, has had a recurrence. In my opinion the syndrome is due to an inflammation of the superior cervical sympathetic ganglion, secondary to the nasophayngitis.

It is as follows: A nasopharyngitis (either alone or associated with involvement of the nose and throat) of a varying degree is accompanied or followed by an earache and a unilateral headache, together with a tender area in the side of the neck over the location of the superior cervical sympathetic ganglion. No complaint is made, however, of the tender area in the neck and it is always necessary to search for this part of the syndrome. Pressure on this area will serve to exaggerate the pain in the head and ear and, if applied at a time when the pain is temporarily absent, it will produce it, the pain then ceasing when the pressure is released; thereby establishing a direction connection between the two.

The pain does not seem to take a definite course as in the case of Sluder's syndrome, nor does it show any tendency to become localized at any particular point. In one case, however, the pain in the side of the head was controlled by cocainization of the sphenopalatine ganglion, but the otalgia remained unchanged. The patient volunteered this specific information following the cocainization without any questions being asked.

Examination of the eardrum always showed it to be free from inflammation and Roentgenograms of the mastoid were likewise negative.

Examination of the nose may be negative, though in one case there was evidence of a bilateral antrum infection. While the in-

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flammation from the nasopharynx in some cases extends into the posterior nares, evidence of sphenoid or posterior ethmoid suppuration was lacking. In the case referred to with a bilateral antrum suppuration, it was not possible to exclude entirely involvement of the anterior ethmoids and frontals.

In searching for the tender area, one must be careful not to mistake an inflamed and enlarged lymph node for the tender superior cervical sympathetic ganglion. This will hardly be possible though, for there would be no effect produced on the pain from pressure on a lymph node. There may be an enlargement of the lymph nodes in this region and very likely will be, but by careful searching the superior cervical sympathetic ganglion will very readily be discovered if it is involved. Slight or moderate pressure only is all that is necessary. If deep pressure were used, pain might easily be produced by the pressure being transmitted along the Eustachian tube to the drum membrane, causing tension upon it. Since the superior cervical sympathetic ganglion lies posterior to and slightly external to the vascular sheath and opposite the body of the second and third cervical vertebra, the pressure should be directed almost directly backward (slightly inward and upward) and just lateral to the pulsating vascular sheath.

To appreciate the ease with which infection may gain access to this region it need only be mentioned that in this space occur such affections as retropharyngeal abscess and other deep cervical abscesses. It is also a well known fact that the removal of an infected foci from the nose or pharynx will often relieve a very annoying and persistent tinnitus aurium. It seems to me quite possible and also quite probable that these cases may be caused by irritation of the superior cervical sympathetic ganglion, secondary to a focus of infection higher up—the infection reaching this region by way of the lymphatics.

The following three cases are more or less typical of the entire affection:

Case 1: F. P., age 36 years, professional man. Came in complaining of a nasopharyngitis. Two days later he returned with pain on the right side of the head and an earache. Examination of the ear failed to reveal any pathology and it was not until two days later that I discovered the tender area in the side of the neck, though I am satisfied now that it was probably present from the beginning. This was my first case and I perhaps overlooked it. The pain and earache lasted in this case for about three weeks. There were periods when it was absent or at least not so annoying,

and as I have said above, during this period I was always able to produce the pain symptoms (headache and earache) by slight pressure over the superior cervical ganglion. Dry heat helped to relieve the pain. The headache and earache persisted fully two weeks after the nasopharyngitis had disappeared.

Case 2: C. I., age 26 years, teacher. Referred by family physician for mastoid operation on account of pain in the left ear and left side of the head of three days' duration. Examination of the nose and throat was negative. Examination of the nasopharynx showed an intense redness in the very roof of the pharynx. Examination of the ear and mastoid failed to reveal any pathology. Palpation of the neck over the area of the cervical ganglion revealed a tenderness and pressure upon this area served to exaggerate the pain in the head and ear. On account of the pain in her head and ear, she had forgotten to mention the pharyngitis to her family physician and it had accordingly been overlooked. A spray of 10 per cent argyrol for the nasopharyngitis was prescribed. Also a 5 gr. tablet of asperin every 2 to 4 hours when necessary for the pain. This case lasted about two weeks and then cleared up entirely.

Case 3: Nurse, age 28 years. Referred to me for a mastoid operation. She had already had a myringotomy done. Examination of the ear was negative except that the incision in the drum could clearly be seen. There was no tenderness over the mastoid. Because of the fact that she, as well as her physician, insisted that she had a mastoid abscess, I had an X-ray picture made. The right ear being the one involved, I had a stereoscopic view made and I also had one made of the left ear for comparison. The X-ray picture was clear cut and very convincing that there was no pathology present. Her headache and earache was quite severe and she had been unable to sleep for two nights. Pressure over the superior cervical ganglion increased her symptoms very markedly. I did not see her again, but was informed by her family physician about three months later that her pain lasted about three weeks and gradually subsided. He also informed me that he had since seen two other similar cases.

THE ATKINSON TRACHEOLARYNGOSCOPE IN EXAMINATION AND SURGERY OF THE LARYNX.

Dr. John F. Callahan, Brockton, Mass.

Examination of the larynx has always been a difficult matter, because no part of the structure except the tip of the epiglottis can be seen by direct vision. For the same reason, operations within the larynx were, until a few years ago, considered impossible. When we realize that Gracia, the first man to see into the larynx with any degree of satisfaction, accomplished this feat not earlier than 1855, we come to appreciate how new a science laryngology is. Gracia was not a physician but a teacher of music, and it was not until long after he had originated his method that anything of practical nature was accomplished. His mirror, which was a crude affair, quite similar in principle to the laryngeal mirror now in use, enabled him to see the vocal cords of his pupils. He wrote a good deal about his discovery from time to time and one of his treatises fell into the hands of Turck, of Vienna, who demonstrated the practicability of the method and introduced it to the profession. His process was not for a long time adopted, however, because of the lack of means for properly illuminating the field. The head mirror, so simple to us now, had not at that time been thought of. Usually the examinations were made by sunlight, and it is manifest that this method could not but be unreliable. Later gas light was used with a reflector held between the teeth, or projecting downward from a spectacle frame. Then came the head mirror, which overcame all these defects of illumination and made indirect laryngoscopy a partial suscess, at least.

As the method became generally adopted a great many obstacles were encountered. A patient with a sensitive throat would often baffle all attempts to see into the larynx because of his constant gagging. This was before the introduction of local anesthesia, without the use of which a large percentage of these examinations could not be made even at this time. Greatly enlarged tonsils were a bar to examinations which were often discontinued rather than disturb organs which were thought to function in the process of digestion, as well as to protect the patient against tuberculosis and other dis-

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eases. The pendent epiglottis was also found to be the cause of frequent trouble and all sorts of instruments were originated to pull it forward. The Burns Pinchette was a favorite device for this purpose. It had never dawned on anyone that a high falsetto note, such as "e" or "ah," would raise the epiglottis sufficiently for the larynx to be seen.

Then, as now, it was recognized, that in many cases the indirect examination of the larynx was not a success even in adults, and that in the cases of children it was nearly always a failure. This realization accounted for the origin of our practical laryngoscopy by the direct method. Kirkstein was probably the first to look directly into

Fig. 1. Instrument in position for introduction with the patient in sitting position.

the larynx through a straight tube, and although, while in his hands this was never made practical, yet it paved the way for numerous other instruments by the use of which, many of the obstructions were surmounted. It is generally agreed that the credit for a method of properly viewing the larynx and operating within it, should be accorded to Killian, who gave the world his celebrated suspension apparatus; since that time his method has been improved upon by Lynch. Chevalier Jackson, with his tubular laryngoscope, has also achieved a task for laryngoscopy that is now recognized throughout the world, as also have Sieffert and Haslinger with their laryngoscope of the bivalve speculum type.

The Atkinson tracheolaryngoscope, which the writer now uses, has without doubt, advantages which are distinctive. In the other instrument mentioned, the construction is such that one blade is superimposed upon the other, the appliances being similar in principle to a bivalve speculum. This type of laryngoscope necessarily may be introduced with less facility than is resultant with the use of the Atkinson instrument. The latter device is unique in the fact that the opposing blade is made in two sections, one of each, when the instrument is closed and ready for introduction, lying besides the spatula blade. This arrangement makes the laryngoscope much less bulky, and almost as easy of introduction as a tongue depressor.

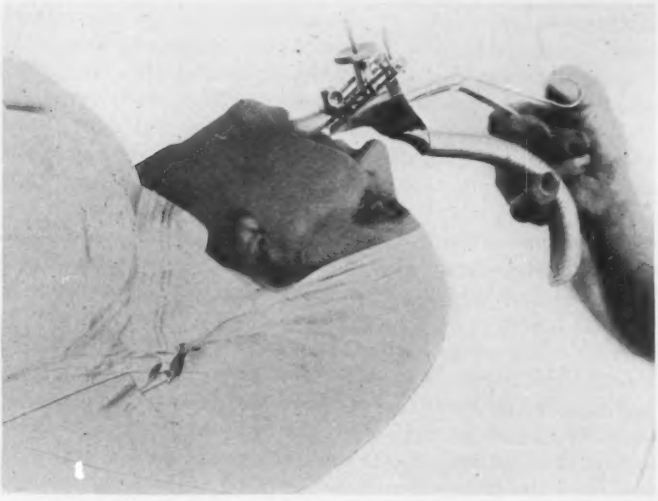

Fig. 2. Patient recumbent. In this position the handle of the instrument is reversed.

The technique of inserting the Atkinson instrument is simplicity itself. If the examination is to be made in the sitting position the patient should sit astride a chair with his chest resting upon its back. With the throat previously prepared with local anesthesia, the distal end of the spatula is made to glide backward for something like an inch behind the dorsum of the tongue. The blade is now slightly elevated, this maneuver bringing the distal end of the spetula just above the tip of the epiglottis. At this point, the tip of the spatula is depressed slightly more as the opposing blade is elevated by a screw, which acts as a lever. This procedure occupies but

a moment's time and the view thus made possible is unusual, due to the construction of the instrument which puts the hinge within the mouth rather than external to it, thereby admitting of a greater spread of the blades than otherwise would be possible. In operating with this speculum, the instrument may be entirely disregarded after it is placed in position, and the handle removed if this is found desirable, for it readily becomes self-supporting; in other words, it retains itself without any help from the surgeon, leaving his hands free to work. True, this modern instrument is radically different, but its simplicity, its ease of operation, and its effectiveness must, I believe, appeal to all laryngologists who use it.

While direct laryngoscopic examination must be considered as an unusually useful innovation, the method necessarily has its limitations. In tractable patients without some special contra-indications existing, it will seldom be necessary to resort to it. Nor will the method ever take the place of the almost daily indirect examination of the larynx now being practiced during routine office treatment. There are types of patients, however, in which indirect laryngoscopy always has been and always will be a failure. In these cases, even with the neurotic, gagging, coughing individuals, direct laryngoscopy, under cocain anesthesia, usually can be employed. Failing in accomplishing this locally, general anesthesia can almost always be administered, and then the examination becomes a very simple and practical matter.

While not even the enthusiast would venture to say that direct laryngoscopic examinations will ever wholly supplant the laryngeal mirror, it is a fact obvious to all who have ever done or even witnessed laryngeal surgery under direct laryngoscopy, that the old method of operating in the larynx by the aid of the will of the wisp picture in the laryngeal mirror, is a thing of the past. The obstacles which interposed when the old method was used were so numerous, the results obtained were so indefinite and unsatisfactory and the number of men attempting surgery by this method were so few, that, in fact, very little of importance was ever accomplished.

However, modern instruments, and preferably, in the author's opinion, the Atkinson model, have so revolutionized laryngoscopy that this formerly formidable type of surgery has now become, in its simplicity, such that an average laryngologist may practice with success.

Barrister's Hall.

OUR PRESENT KNOWLEDGE OF PROCAIN WITH A BRIEF REPORT ON EXPERIMENTAL CAROTID INJECTION OF THE DRUG.*

DR. KARL MUSSER HOUSER,** Philadelphia.

It is always of value for the surgeon to possess all available knowledge about the materials which he is obliged frequently or occasionally to use. Quite often this information can be obtained only by a careful and painstaking search of the literature, which requires more time than the busy operator can possibly give. To the otolaryngologist the use of procain is a necessity and it is the purpose of this article to make available for him in compressed form our present knowledge of the powers, dangers, and limitations of this drug.

History. In 1905, Einhorn[1] produced and Braun[2] tested a stable, non-irritating, slightly toxic compound capable of producing temporary anesthesia of sensory nervous structure. Soon after, Liebl,[3] in personal subcutaneous injections of this substance produced no toxic symptoms with 0.4 gms. (6 grains) and a very slight reaction with 0.7 gms. (10½ grains). This drug, which chemically was para amido benzoyl diethyl amido ethanol, later became known and marketed under the trade name "novocain." Previous to the world war novocain could be procured only through German firms, which exercised a monopoly over its manufacture and sale. The Trading with the Enemy Act passed by Congress following the severance of diplomatic relations with Germany gave to the Federal Trade Commission the control and disposal of enemy-owned patents. This provision made possible the production of patented foreign products by authorized firms in the United States. The original trade name, however, could not be used, and on this account the synonym "procain" was adopted by various producers in this country. In France, products said to be identical, which were termed neocain and syncain, were produced under similar governmental regulations.

Chemistry. Procain[4] occurs as a white crystalline substance soluble in one part of water and thirty parts of alcohol. The reaction of an aqueous solution is neutral. From such solutions it is readily precipitated by the common alkaloidal reagents. Possessing a melt-

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*From the Department of Otolaryngology, University of Pennsylvania.

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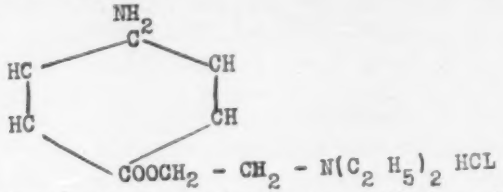
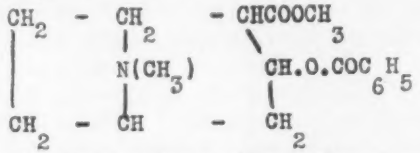
ing point of 156° C., it is quite stable and is not decomposed at the temperature of boiling water. Chemically, it is termed para amido benzoyl diethyl amido ethanol and is represented by the following structural formula:



It is interesting to compare the structure of this drug with that of cocain, the chemical name of which is benzoyl methyl ecognine and which structurally is:



It has long been known that the latter substance can be broken up readily by heating in aqueous solution into methyl alcohol and benzoyl ecognine. The investigations of Stockman[5] and Filehne[6] gave rise to the idea that the benzoyl radicle was the essential factor in imparting anesthetic properties to cocain. Poulsson,[7] in 1890, stated that esterification of benzoyl compounds with aliphatic radicles was the most important step in producing compounds possessing the power of local anesthesia. Since then many such derivates of benzoic acid, C_6H_5COOH, which possess such properties, have been prepared. Inspection of the structural formula of procain shows its close relation to benzoic acid. In addition a comparison of that formula with that of cocaine shows both to contain the benzoyl radicle and an alkamine ester. Otherwise, there is no noteworthy structural relationship.

Present Usage. The use of procain at present is practically universal. It is used by the surgeon in operations on all parts of the body. Appendectomies, thorocotomies, amputations, hernorrhaphies, fracture reductions, cholycystectomies, tonsillectomies, thyroidectomies, mastoidectomies and countless other surgical procedures have all been rendered painless by means of this substance. The margin of safety must be high for fatal results that can be directly traced to the drug are comparatively few. The general surgeon thinks little of possible procain toxemia and uses the drug *ad libitum*. Bartlett[8]

has used as much as 1000 c.c. of ½ per cent solution in infiltration anesthesia without serious toxic symptoms. This is equivalent to 5 gms., or 75 grs. of procain. Sharpe[9] reports a series of 459 major surgical procedures under local procain anesthesia. He uses as much as 360 c.c. (12 fluid ounces) of ½ to ¼ per cent procain with varying amounts of 1:1000 adrenalin chlorid solution. No untoward results or symptoms are recorded. Some surgeons routinely inject 0.15 to 0.10 gm. (2½ to 1½ gr.) into the spinal canal.

Eastman, Erdman and Bonn[10] state that less than 2 gm. in individuals not susceptible gives an adequate margin of safety. Mayer,[11] who is chairman of the Committee of the American Medical Association for the Investigation of Local Anesthetics, has come to believe that in most of the deaths resported as due to procain, some cocain, too, has been used. In spite of all this evidence of the safety of procain use, it must not be forgotten that we are dealing with a poison which has lethal properties and a lethal dose for each individual.

Toxicity. Much difference of opinion exists concerning the toxicity for man. Halsey[12] considers it to be one-half as toxic as cocain. Cushney,[13] one-third; LeBrocq,[14] one-half; Meyer and Gotlieb,[15] one-third; Marshall,[16] one-third, and Biberfield,[17] one-sixth. These wide variations are probably the result of different methods in making experimental observations without having constant the factors, speed of injection, and solution concentration. These factors have been demonstrated to be of great importance. In fact, certain observers state that the toxicity for rabbits when injected intravenously varies directly as the speed of injection and solution concentration.[18]

There is little doubt that some patients are more susceptible than others. Fatal results have occurred following the use of small amounts of the drug. The injection of as small an amount as one grain has been followed by death.[19] Although no definite experimental explanation of this condition has been given, it is generally admitted that in such patients some abnormality of the respiratory center and heart has made them especially sensitive to the drug. A preliminary test for such hypersusceptibility is always advisable, especially when the surgeon is working in highly vascular tissue where absorption is rapid. This can easily be performed by the injection of ½ c.m. of a ½ per cent solution and allowing five minutes to elapse before proceeding further.

The value of this maneuver is perhaps best illustrated by a recent experience of the author. A young adult female, suffering from pulmonary tuberculosis, was referred to him for tonsillectomy under

local anesthesia. The tonsils were small, but decidedly septic and removal was considered advisable by her internist. One hour before operation she was given 1/6 gr. of morphin sulphate hypodermically. On admission to the operating room she showed signs of apprehension, but nothing of an alarming nature. She was given, according to routine, the usual ½ c.c.m. submucosal, pre-operative injection of ½ per cent procain solution at the superior pole of the right tonsil. Almost immediately she collapsed. The radial pulse was imperceptible, respirations were so shallow that their detection was difficult, her appearance was ghastly, unconsciousness was total, and it was difficult to determine immediately if she still lived. No convulsive movements of any kind occurred. The patient was placed in a supine position on the floor of the operating room with the body higher than the head. An adrenalin hypodermic was ordered and artificial respiration started. Before the hypodermic could be given the pulse became perceptible, respirations returned to normal and consciousness was restored.

The test dose had been so small that the operator felt certain the attack had been one of syncope rather than procain toxemia and it was decided to continue the operation after the administration of another test dose with the patient in a recumbent position. Another ½ c.c.m. of the procain solution was given to the patient lying upon an operating table. After five minutes no toxic symptoms had appeared. Sufficient anesthetic was then injected and the operation completed in the usual manner. No further alarming symptoms developed.

In this case had the total dose been given immediately almost certainly the alarming symptoms would have been interpreted as those of procain poisoning. Had death occurred, even with this small dose, procain would have been given the credit of the fatality. The subsequent completion of the operation with procain as the anesthetic conclusively proves that in this case no hypersuceptibility to the drug existed.

Physiological Action. Valuable information concerning the action of procain and its fate in the body can be gained from the work of Roth[20] and that of Eggleston and Hatcher.[21] Roth experimented with frogs, mice, rats, guinea pigs, and rabbits to determine the action of both procain and cocain upon the intact animal. In addition the action upon smooth muscle and blood pressure was tested. In cold blooded animals he found that the former was slightly more toxic than the latter. In all warm blooded animals, tested cocain was at least five times as toxic as procain when injected subcutaneously. If injected intravenously into rabbits, the toxic action of

cocain was four times greater than procain. He, too, found that the manifestations of poisoning depended greatly upon the speed of injection and the concentration of the solution used.

In dogs, cats and rabbits the effects of cocain and procain on blood pressure and respiration are essentially alike. Small doses produce a slight rise of blood pressure, accompanied by respiratory stimulation. Large doses quickly given produce an abrupt fall of pressure. Roth found that death from procain when a lethal dose was given slowly, was always an asphyxia due to paralysis of the respiratory center, whereas if a large lethal dose was given suddenly cardiac arrest occurred simultaneously with or preceding, the respiratory failure.

The symptoms of poisoning in cats are usually inaugurated by a pronounced stimulation of respiration followed by an abrupt fall in blood pressure. If the dose is severely toxic, but not lethal, the heart, which was first slowed, in about a minute begins to improve. Later blood pressure rises and respiration becomes normal. One or more clonic convulsions may occur. In rabbits[21] when a lethal dose is given, respiration is first stimulated, which is followed by struggling movements and clonic convulsions. Breathing later becomes shallow, air hunger marked, and respiration ceases before the heart beat.

The facts derived from the work of Eggleston and Hatcher[21] are extremely valuable and interesting. They found that if a fatal dose of procain was injected very slowly intravenously one fatal dose was destroyed every twenty minutes. In other words, a fatal dose could be given intravenously every twenty minutes, provided it was given slowly. In this manner sometimes as much as nine times the usual fatal dose could be given without fatal results.

With cocain, however, an hour was necessary before a fatal dose was detoxified. Subcutaneously, five times the fatal intravenous dose could be injected without causing death. In the cat they found that solutions were rapidly destroyed and rendered inert by perfusion through the liver. From these observations it is reasonable to conclude that procain is much more readily rendered non-toxic after admission to the blood stream than are equally toxic quantities of cocain and that the liver is an important factor in this detoxification.

Adrenalin and Procain. Almost every surgeon who uses procain uses it in combination with adrenalin chlorid solution, varying in strength from 1:10,000 to 1:100,000. The action of this solution by local vasoconstriction delays absorption with a resultant prolonged anesthesia. Hemorrhage is less likely to be severe. Some observers[10] have noted that the addition of adrenalin to procain

solutions increases the toxicity 2 3/5 times when tested intravenously on rabbits. In spite of this fact, it seems advisable to use adrenalin chlorid solution in a 1:50,000 concentration, as advised by Sollman,[23] for the advantage of the delayed absorption offsets any increase in toxicity provided the injection is not made directly into the blood stream.

It seems to be the opinion of all observers who have tested the anesthetic properties of procain that by no means does its anesthetic efficiency vary directly as its percentage concentration. In other words, it is not necessary to use high concentrations to secure perfect sensory paralysis. One-half per cent is sufficient in the opinion of most surgeons to give adequate infiltration anesthesia.

It was demonstrated by Meeker and Frazer[24] that the lethal dose of procain for experimental animals varied considerably when injection was made into the blood stream by various routes, i. e., arterial or venous. In all peripheral veins the lethal dose was the same. When injection was made into the femoral artery, however, the lethal dose was much greater than when injection was made into the common carotid. It was the feeling of these observers that since most procain fatalities occur in head and neck surgery that they could be due perhaps to direct injection into the vertebral or internal carotid arteries, resulting in the higher centers receiving a highly concentrated procain dosage with a consequent immediate paralysis.

Author's Investigation: The author, working with Dr. George Wagner, of the Department of Surgical Research of the University of Pennsylvania, experimented with several dogs in the attempt to learn whether a dose of procain larger than that usually used in tonsillectomy would produce death if injected into the internal carotid artery. It is conceivable that this artery could receive the total dosage at the time of injection.

The first dog was subjected to ether anesthesia, the common carotid exposed, and its branches, including the external carotid, ligated. Injection of 2 c.c.m. of a 1 per cent cocain solution containing adrenalin chlorid (1:20,000) was then performed at 3 minute intervals for four doses. A total amount of 0.12 gms. of procain with 0.0024 gms. of adrenalin chlorid were given (1.8 grs. procain and 0.0036 grs. adrenalin). The respirations became more rapid and shallow, but soon returned to normal. In 30 minutes the dog had completely recovered and was in the first stage of ether anesthesia. With the second dog the carotid was prepared as before. Following this he was allowed to recover from the anesthetic, when the ex-

periment was repeated. The results were not different from those previously obtained.

Although these experiments are not sufficiently complete to make sweeping deductions, they do show that more than twice the amount of procain used on one side in the usual tonsil operation can be injected directly into the internal carotid artery of the dog without giving rise to severe toxic symptoms.

Precautions and Therapeutic Measures. The knowledge of procain action we now possess should enable us to intelligently formulate some precautionary measures for the prevention of poisoning and to decide upon the remedies to apply should toxic symptoms arise.

1. The surgeon should be on guard for susceptible individuals and detect them by a preliminary subcutaneous dose of ½ c.c.m. of a ½ per cent solution of procain.

2. He should use the lowest concentration of procain sufficient to produce anesthesia; this is generally admitted to be ½ per cent.

3. He should insist upon using a carefully prepared solution for injection. Too often the solutions found in our hospital operating rooms are of questionable age and careful examination detects the presence of a sediment in the flask. It is much safer to use sealed ampules for each operation, discarding that amount of solution which has not been used.

The question of the deterioration of procain solutions has been investigated by Bonar,[24] who found that if a solution is sterile and sealed no change occurs in its anesthetic properties or toxic action in several months. The yellow tinge sometimes seen in these solutions is apparently an oxidative process which does not affect the action of the drug. The sediment sometimes seen is due to a mould contamination which condemns the solution.

4. Injection should not be made directly into the blood stream. This can best be avoided by the use of a glass syringe which is fitted with a plunger on the end of which is a ring for the thumb. By means of this negative pressure can be made. When this is done, if the point of the needle has entered a blood vessel, blood immediately appears in the syringe.

5. Rapid absorption can best be prevented by using adrenalin chlorid in a concentration of 1:50,000 incorporated with the procain solution.

With regard to remedies, once poisoning has occurred, it is worth while to bear in mind the work of Eggleston and Hatcher.[21] In their experiments they gave to a series of 18 cats, 117 per cent to

200 per cent of the lethal dose of procain and attempted resuscitation by means of artificial respiration and cardiac massage. In only two instances were they able to revive the animals. In another series when similar doses were given, but where adrenalin cholorid intravenously was used in addition to artificial respiration and cardiac massage, they were usually able to resuscitate the poisoned cat. Caffein and atropin had no effect in reducing the toxicity of the drug. Ouabain, an alkaloid of digitallis, given 15 minutes to several hours before the procain, protected cats from 117 per cent to 175 per cent of the lethal dose.

The therapeutic indications, therefore, in cases of poisoning apparently are to continue respiration and support circulation until the liver and other organs which may share in the disintegration of procain be given an opportunity to exert their detoxifying actions, which in the case of procain is rather rapid. To best accomplish these things artificial respiration, cardiac massage, and adrenalin chlorid intravenously or directly into the heart should be used (10 to 15 minims.). The experimental results with ouabain indicate that the drugs of the digitallis series are of value in combatting the toxic results if given pre-operatively. Whether similar protection could be obtained by administration after poisoning has occurred is not known, but in severe cases it should be worth a trial.

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*The author wishes to thank Dr. J. E. Sweet, of the Department of Surgical Research of the University of Pennsylvania for helpful suggestions and the use of his laboratory.

Physicians Building.

A SIMPLE NASAL SUCTION DOUCHE.*

DR. A. J. SHEKTER, Jamaica, N. Y.

The apparatus illustrated is intended for the home use of the patient; it is simple in its makeup and inexpensive. It does not depend for its operation upon overhead waterbag, water or electric pump. It is compact and does all that is expected of a negative pressure suction irrigator. All parts are made of noncorroding material easily taken apart and sterilized by boiling. Valves are large and so built as to prevent clogging from nasal discharges. The suction bulb is of a special make and by its thickness and rib construction will "draw" quickly when released. It will produce a sufficient negative pressure to draw pus and infected matter from the nasal cavities and sinuses of the average case and at the same time draw up a column of irrigating fluid to wash these discharges away.

As stated above, it is intended primarily for the home use by the patient in conjunction with the regular office treatment by the specialist. It is an improvement on the various nasal irrigators already on the market, with which the writer is familiar, because of its simplicity and efficiency, being really autosuction. It is intermittent in its action and does not overcongest the parts. Since the suction created in this apparatus is produced by means of a negative pressure hand bulb, the negative pressure thus formed is intermittent and it is this intermittency which is one of the essential factors in withdrawing discharges from the cavities. It will be found to give good results in the types of nasal and sinus diseases where suction, irrigation and hyperemia are used for therapeutic measures.

The nasal douche is made up as follows: *A*. A glass nasal tip so constructed as to permit quick and direct return flow; *B*. rubber tubing of two colors, white and red; *C*. rubber bulb (especially constructed for negative pressure); *D*. valves and end weight.

*Presented at A. M. A. Convention, Atlantic City, May 20, 1925, Section Laryngology, Otology and Rhinology.

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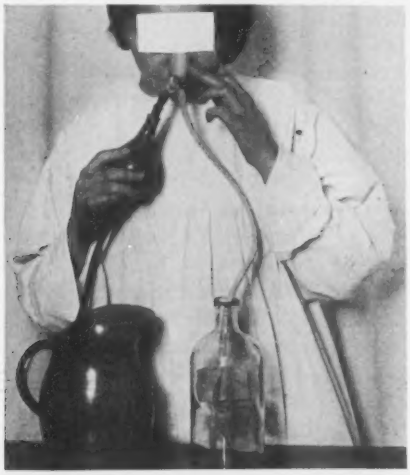
The procedure for its use is the same as for any negative pressure apparatus. Patient places end of white rubber tubing into irrigating fluid; the end of the red rubber tubing is placed in a waste basin or dish partly filled with water to cover the end valve.

By grasping the bulb, the nasal piece is placed into the nostril to be irrigated and the other nostril closed with the index finger of the free hand as shown in the sketch. Repeating the letter "K" and at the same time repeatedly squeezing the rubber bulb, a sufficient negative pressure is produced in the nasal cavities and sinuses to cause an evacuation of the discharge, which in turn is quickly washed



away and sucked into the waste jar by the irrigating fluid drawn up. This action takes place in a milking-like intermittent manner.

The amount of negative pressure in the nose can be controlled by the frequency and force exerted on the rubber bulb and since the force of irrigation is never greater than the amount of this negative pressure there can be no danger of forcing infected material into the middle ears.

I am sure this apparatus will be found a valuable aid for the home treatment by the patient in conjunction with the regular office treatment by the specialist. This irrigator is made by Wregg, Inc., 87 Lexington avenue, New York City.

DEMONSTRATION OF A NEW INSTRUMENT FOR THE TREATMENT OF O.M.C.C. WITH OVERHEATED AIR.*

DR. JOHN GUTTMAN, New York City.

About 50 per cent of the patients visiting the office of an otologist complain of diminished hearing and noises in the ear, suffering from O.M.C.C., otosclerosis, or some other form of an affection of the sound-conducting apparatus.

The treatment of these cases is confined almost entirely to catheterization or Politzerization. In exceptional cases medicated air or fluid is insufflated into the tympanic cavity. A few attempts were also made by J. A. Andrews,[1] G. W. Hopkins,[2] and others to insufflate heated air. Owing to the difficulty in the manipulation of these different procedures and lack of beneficial results, these methods of treatment, with the exception of catheterization and Politzerization, do not find much favor with the otologists.

The instrument recommended by the writer is designed to heat air coming out from a Politzer bag or compressed air pump. This air can be heated to about 200° F., provided the pressure is kept constant. A decrease of pressure diminishes the degree of heat.

The instrument consists of the following parts: *1.* A closely wound copper tubing; *2.* an electric heating unit enclosing the above; *3.* a heat regulating device; *4.* an enclosing insulated shell with intake and outlet for the air current.

These parts are built into a nickel-plated metal shell 2 inches in diameter and 5 inches long. Both ends of the coiled inner tube lead to the outside of this shell.

The air intake tube is provided with a hose nipple over which a rubber tube can be slipped to connect the apparatus with the air supply. The outlet tube has on its extreme end a hose nipple to which a 3/16-in. rubber hose can be attached. This in turn connects with an ear catheter.

It is necessary to make the hose line from the apparatus to the catheter as short as possible, so as to avoid the loss of heat through radiation.

In order to avoid the contact of the heated metal catheter with the tip of the nose which is very sensitive to heat a thin rubber tubing can be slipped over the distal end of the catheter.

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*Read before the Otological Section of the N. Y. Academy of Medicine, Jan. 8, 1926.

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The heat regulating part consists of a thermostat. This controls the heat by turning a large knurled head screw. Turning the screw to the right will increase the heat and turning it to the left will decrease it.

The apparatus may be operated from any 110-125 volt lamp socket.

The instrument can be held in the hand or can be clamped to a stand, thus leaving one hand free for action.**

The manipulation of the instrument is quite simple, and the reason of its use is obvious.

In inflammatory joint affections, especially in cases of ankylosis and pseudo membrane formation in the capsule of the joint, the use of heat in the form of hot packs and electric heat bath comprise a very efficient and popular form of treatment, as a change in the blood circulation produced by the heat facilitates the absorption of the exudate.

The pathological changes in the majority of cases of O.M.C.C. consist in the formation of an exudate, which in course of time becomes organized, forming pseudo membranes, adhesions, and ankylosis of the ossicles.

Is it not reasonable to expect, that superheated air insufflated through a catheter into the tympanic cavity by the demonstrated electrical instrument will also cause a change in the blood circulation by an increased flow of blood and a readier absorption of the exudate?

Following this treatment we can see in a majority of the cases an increased redness, especially in the upper quadrant of the tympanic membrane. Fine, thin blood vessels will be seen running down towards the short process of the hammer and along the upper and anterior border of the tympanic ring. The effect of this treatment is in suitable cases quite striking. In a comparatively short time the noises in the ear become less distressing, and in some cases disappear entirely. The beneficial effect upon the hearing is in some cases also quite marked. Up to the present the effect of this treatment upon the hearing was not examined in a sufficient number of cases with my acumeter (audiometer), and therefore definite, reliable statistics upon its effect on hearing cannot be presented.

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987 Madison Avenue.

**The instrument is made by the Klett Manufacturing Company, 202 E. 46th street, New York City.

ACUTE OTITIS MEDIA IN ADULTS.

Dr. Louis K. Pitman, New York.

In the treatment of acute otitis, it is the aim of the otologist to ward off complications, and this may be best effected by treating the disease adequately in its early stages. If the disease progresses in spite of these efforts, then systematic treatment should be resorted to. In a previous issue of The Laryngoscope I described the eustacheter, now called "Catheterizing Eustachoscope," an instrument for treating various middle ear and Eustachian tube diseases. I found it to be very helpful in this instance—in checking the development of acute otitis as well as in the treatment of later stages. In order to show how it may be used to the best advantage, a review of acute otitis and its treatment as the disease progresses is necessary.

The organism in consideration here is either the noncapsulated coccus (streptococcus) or capsulated (diplococcus, or streptococcus mucosa). Before the tympanic membrane is broken, it is always a monobacterial infection, *viz.*, streptococcus, diplococcus, or streptococcus mucosa. When the membrane ruptures, the staphylococcus makes its entrance, a secondary infection resulting, such as streptococcus and staphylococcus, diplococcus and staphylococcus, or streptococcus mucosa and staphylococcus. It is very essential for the otologist to make a bacterial investigation to aid him in his diagnosis and prognosis.

The most reliable results are obtained from the paracentesis knife following a paracentesis through an intact drum membrane, the external auditory canal having previously been sterilized. A culture is prepared, from which three smears are made. The first is stained with methylene blue; the second with carbo fuchsin; the third with a fresh water solution of thionin. The methylene blue stains the common streptococcus; the carbo fuchsin, the diplococcus; the thionin, the streptococcus mucosa.

I. THE NONCAPSULATED (UNCOMPLICATED) OTITIS.

This disease begins with pain in the ear, which increases in severity for a few days.

The first stage, or stage of serous secretion. The drum picture shows reddening over the malleus, including the Shrapnell's mem-

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brane, but through this redness the short process and the handle of the malleus are still visible. This redness is usually confused with Tröltsch's stripes artificially brought about by the movement of the speculum. The Tröltsch's stripes are always behind Shrapnell's membrane, while an acute otitis is redness in Shrapnell's membrane. In a short time the drum picture changes: the entire tympanic membrane becomes red, and the malleus is no longer visible except for the short process. The lining mucosa of the middle ear and the Eustachian tube becomes swollen, and finally an occlusion of the latter results. This is the point where the secretion begins, and it is here that an attempt should be made to check the progress of the disease.

To this end, I use the eustacheter, which enables me to introduce a No. 4 or No. 5 special kidney catheter that I have adapted to this work, into the entire length of the Eustachian tube or as far as the isthmus. The catheter here has a three-fold purpose: *1*. By its very presence it acts as a mechanical dilator of the Eustachian tube. *2*. Through the lumen of the catheter I inject with the aid of a hypodermic syringe a small amount of 10 per cent cocain diluted with adrenalin, thus increasing the dilatation. *3*. The catheter is still kept in place, and after five minutes I inject ½ c.c. of ½ per cent silver nitrate for treatment. Care should be taken not to inject any fluid into the middle ear, but to be limited to the Eustachian tube canal. This end is accomplished by having the outlets of the catheter at its side and not at the tip, and injecting while the patient's head is in the upright position. This will allow the excess to flow back into the nasopharynx. (See Fig. 1 and Fig. 3.) The catheter is now removed. I also find it helpful to drop into the external ear some carbolglycerin (3 per cent).

Early experimentation in checking the progress of the disease by this method has been rather encouraging. If the attempt to check the disease has not been successful, nothing has been lost. On the contrary, the mere dilatation of the Eustachian tube in itself militates against possible future complications.

If the disease progresses, the drum membrane changes. The short process is no longer visible, the drum is red and bulging, and the drum membrane is covered with white spots (desquamated epithelium). In this stage, the serous secretion is behind the drum membrane, and at times some of the secretion fights its way out into the external meatus through the interstitial tissue of the drum membrane. It is also at this point that the serous exudate is changing

to purulent. Now a paracentesis is necessary, as well as a repetition of dilating and treating the Eustachian tube as described above.

Second stage, or stage of purulent secretion. Here the drum membrane is broken, and through the opening is seen oozing out thick, purulent secretion. In this stage the patient is relieved of pain, but complains of deafness, fullness and pulsations. The treatment calls for a continuation of dilating and treating the Eustachian

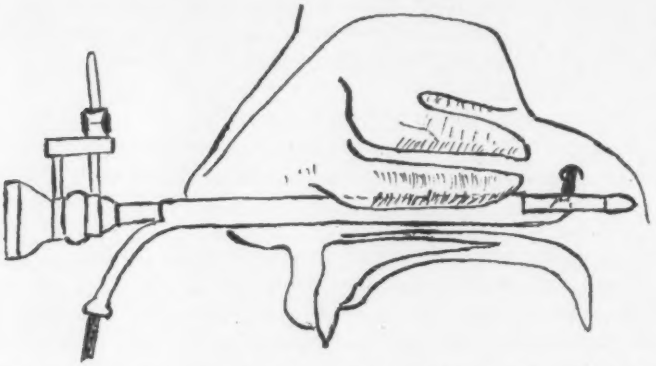

Fig. 1. Eustacheter introduced and catheter entering Eustachian tube.

Fig. 2.

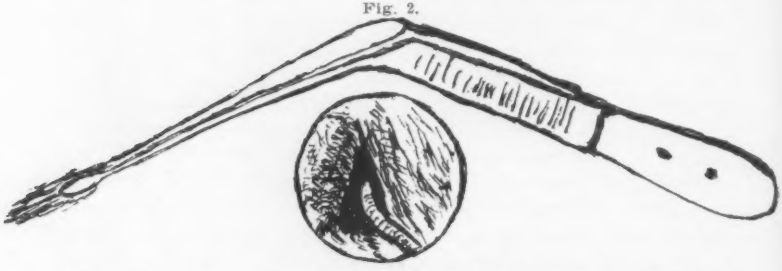

Fig. 3. Catheter entering Eustachian orifice as seen through nasopharyngoscope.

tube twice weekly, as described in the first stage. The external meatus is to be kept clean by irrigating when necessary with hydrogen peroxid diluted with three parts warm water. A 5 per cent silver nitrate applied twice a week by means of a cotton applicator to the external ear will prevent the formation of an external otitis. A wooden applicator is undesirable because it blocks one's vision,

and blind application does more harm than good. I recommend a small ear forceps grasping at its end a small piece of cotton previously rolled in the fingers (Fig. 2). After flaming, the forceps are ready for use. The duration of this stage is about two weeks.

Third stage, or mucopurulent stage. The secretion has changed to mucopurulent. In this stage the patient usually complains of deafness, feeling of fullness and tinnitus. To overcome these symptoms, the Vienna clinics resort to air inflation (Politzerization or catheterization), but the objection to this procedure is that it may blow infected particles into the antrum, and serious complications may follow. However, if my method of Eustachian tube dilatation and treatment is used during the progress of the disease, the patient does not complain very much of the symptoms mentioned above. In fact, he may be entirely oblivious of this stage. My treatment here continues that of stage two. The duration of the third stage is about two weeks.

Fourth stage, or catarrhal stage. The secretion has stopped, the drum perforation has healed, the catarrhal symptoms are slight. The treatment is still dilatation. The duration of this stage is about ten days.

The disease thus runs its entire course in from five to six weeks.

II. MUCOSUS OTITIS (COMPLICATED).

This disease is caused by capsulated cocci—streptococcus mucosus, or diplococcus.

It is not my aim here to dwell in detail upon this form of otitis, since it does not respond well to local attention and surgical intercession is necessary. I shall follow the outline of Prof. E. Ruttin, of Vienna, which may help the otologist to detect this form of otitis, for an early recognition makes the prognosis less grave.

First stage. The only symptom is a slight, sticking pain, but often overlooked by the patient. This stage is of brief duration.

Second stage, or latent stage. The patient does not complain of pain, but of general fatigue. The hearing is poor and not improved by Eustachian tube dilatation (thus differentiating it from a serous catarrhal condition), and tinnitus is present. The drum picture shows a grayish-red membrane, not transparent, the details are visible but a little veiled. This stage may last for months, with the ear not discharging except that at times the drum perforates itself, discharging a bit and then closing again. When doing a paracentesis, the sensation is that of cutting into bacon, as Prof. Ruttin says. This is due to the fact that the capsulated cocci produce a

special form of connective tissue, thus filling the entire tympanic cavity with a pillow of connective tissue. This same tissue is found in the mastoid bone when operating on a complicated mastoiditis.

Third stage, or intracranial complications. The organism slowly destroys the bone until it reaches the sinus or dura, or both, causing serious complications. It is therefore to the interest of the otologist to do a paracentesis at the second stage when the presence of mucosus otitis is suspected, and to culture and stain the organism. The treatment is an immediate mastoid operation with the wound exposed, so that the otologist may effect an entrance at some future time should it be necessary.

221 West 112th Street.

A NEW KNOT TIER USED IN CONTROLLING TONSIL HEMORRHAGE.

DR. CARL DAVIS RENDER, Chicago.

The above instrument is used by introducing the index finger into the thimble-like attachment and using the long extension with slot in end to shove the knot home. It can be used on either hand and is so made that it will fit practically any size finger.

Its advantages are:

1. It leaves three fingers and the thumb free to hold the loose end of ligature while shoving the knot home.

2. In doing local tonsils it produces no gagging if the extension is prevented from touching the pharyngeal wall (a matter which is not difficult to do). While the introduction of the fingers into the mouth or throat invariably produces gagging.

3. It does not require the aid of an assistant.

Wrigley Building.

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NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLOGY.

Regular Meeting, Feb. 12, 1926.

SYMPOSIUM ON PAPILLEDEMA.

a. **Papilledema from the Viewpoint of the Otologist.** Dr. Edward B. Dench.

b. **Papilledema from the Viewpoint of the Ophthalmologist.** Dr. Conrad Berens.

c. **Papilledema from the Viewpoint of the Rhinologist.** Dr. E. Ross Faulkner.

d. **Papilledema from the Viewpoint of the Neurological Surgeon (Author's Abstract).** Dr. William Sharpe.

Papilledema from the Viewpoint of the Otologist. Dr. Edward B. Dench.

I wish to preface the few remarks which I make tonight by the statement that I shall discuss the changes occurring in optic papilledema simply from the viewpoint of the otologist. Unfortunately, I know little about the microscopic anatomy of the various conditions of the fundus, neither do I know the finer physical differentiations between the simple edema of the disc and an edema combined with an inflammation of the filaments of the optic nerve, in other words, an optic neuritis or neuroretinitis as distinguished from a simple swelling of the optic disc. Unfortunately, I have not used the ophthalmoscope myself for many years and have always depended upon my ophthalmological confreres to give me the fundus findings in all cases. This will explain my ignorance of the more technical terms which are used concerning the various conditions.

In the first place changes in the fundus oculi may occur as the result of any intracranial condition which gives rise to increased intracranial pressure. We are concerned tonight simply with those cases in which the intracranial pressure is increased by some otitic condition. Although it is my impression that one or two cases have been reported in which a simple inflammation of the mastoid without any intracranial complication was responsible for a papilledema, or at least for some abnormality in the optic disc, I myself have never seen such a condition. Perhaps if the fundus oculi were examined in each case of mastoid involvement, and this I think would be a very wise and valuable procedure, it is possible that changes in the ocular fundus might be found. In my own experience, however, I have never observed a case of papilledema complicating a middle ear condition unless some intracranial condition were present. It should be stated further that any of the intracranial complications of middle ear suppuration may be accompanied by certain changes in the optic disc. These changes may vary from a slight engorgement and tortuousity of the veins to an actual swelling of the head of the optic nerve, and blurring of the margins of the disc. Under certain conditions an actual neuritis of the optic nerve takes place.

The first change, that is, an increased tortuousity and dilatation of the veins with blurring of the margins of the disc, is the result of increased intracranial pressure. Such a fundus change may occur as a result of any intracranial complication from any cause, and all of the intracranial complications of middle ear suppuration may cause these changes to a varying degree. The intracranial middle ear complication least likely to cause a papilledema is an extradural abscess, that is, a collection of pus between the dura and the calvarium. This, the simplest and least dangerous of the intracranial complications, may cause changes in the appearance of the optic disc, but rather infrequently. It should be understood that while as a rule if these

optic disc changes are unilateral they usually occur on the side of the ear lesion, this rule is by no means absolute. I have seen cases in which the optic disc of the affected side was perfectly normal, and the opposite disc showed a marked papilledema. This applies, of course, not only to cases of extradural abscess, the simplest of intracranial complications of middle ear suppuration, but to all of the other intracranial complications of this disease as well. Sinus thrombosis, otitic meningitis, and both cerebral and cerebellar abscess are all prone to produce changes in the optic disc.

Without going into the exact figures, I should say that optic disc changes were more constant in meningitis than in any of the other intracranial complications of otitic origin. They occur, very frequently, however, in brain abscess, both cerebral and cerebellar abscesses and also in cases of sinus thrombosis. In the cases of brain abscess and of meningitis, the general condition of the patient is frequently so desperate that the condition of the fundus oculi is simply sought as a corroborative diagnostic measure. In cases of sinus thrombosis, however, the general condition of the patient is not so serious and the fact that these changes have taken place in the optic disc is important not only because they constitute an important diagnostic feature of an intracranial condition, but also because the effect of the condition on the subsequent vision of the patient has to be considered. I think that I can safely say in the majority of cases where we have had a sinus thrombosis necessitating either ligation or excision of the internal jugular vein that certain changes have been present in the optic disc on one or both sides. If the condition is allowed to persist there is danger of serious impairment of vision.

In these cases repeated lumbar punctures have, at my hands, been entirely successful in relieving the increased intracranial pressure and in all of these cases the vision became normal. Repeated lumbar puncture, then, has in my hands been the best method in preserving vision in cases of optic nerve changes following intracranial complications. While, of course, in brain abscess, evacuation of the abscess ordinarily relieves the intracranial pressure to such a degree that optic nerve changes retrogress rapidly, in their persistence lumbar puncture is of undoubted value, and the same applies to cases of meningitis.

Neoplasms either above or below the tentorium may give rise to certain disturbances of the eighth nerve, bringing these cases under the observance of the otologist. Here the association of certain aural conditions with signs of intracranial pressure are of great value in confirming the diagnosis in doubtful cases. The same applies to certain aural conditions following constitutional diseases, such as lues, diabetes, arteriosclerosis and others.

The point that it seems to me more important to bring out is that even slight changes in intracranial pressure are sufficient to cause marked changes in the ocular fundus. In one case reported by me a number of years ago, in which following an acute middle ear suppuration the Gasserian ganglion was freed from dural adhesions, I introduced a folded strip of rubber tissue to the apex of the petrous pyramid. The fundus oculi had previously been normal. In twenty-four hours after the introduction of the folded rubber tissue, there were distinct evidences of a papilledema. This disappeared in twenty-four hours after the removal of the rubber tissue. If the very slightly increased intracranial pressure incident to the introduction of a rubber tissue drain could cause such rapid changes in the fundus oculi, it is no wonder that pathological conditions, either of an inflammatory, circulatory or a neoplastic nature will give rise to similar conditions.

The changes which take place in the fundus then are of a two-fold nature. First, those which are the direct result of increased intracranial pressure, that is, changes due to a mechanical cause, pure and simple, and also changes which are of an inflammatory nature, that is, those in which we have an actual neuritis of the optic nerve fibers. There is no reason as far as I can see why these two conditions may not coexist, that is, they may have a papilledema due to pressure and in addition to this have certain changes in the nerve itself, due to inflammatory changes within the cranium, that is, a neuroretinitis. In a recent case of sinus thrombosis operated upon in which

an extradural abscess was found at the time of operation, the examining ophthalmologist pointed out quite clearly that in one eye we had a simple papilledema, while upon the opposite side there were in addition to a papilledema certain evidences of a neuritis of the optic nerve. He even went so far as to suggest an accompanying meningitis, although the patient had no symptoms of meningitis. I explained to him that the only area of dura exposed was a small area over the lateral sinus, yet to his mind this was quite sufficient to have caused the inflammatory changes in the optic nerve itself.

The records of my own cases have been incomplete as far as this is concerned, and I cannot say whether in all cases of meningitis, inflammatory changes in the optic nerve are present or not. I do know, however, that in a great majority of these cases we have a papilledema whether or not the inflammatory changes are present. In the constitutional diseases mentioned above, lues, diabetes, nephritis and arteriosclerosis, the optic disc change is not limited to a papilledema. In fact, in many of these diseases this condition may be wanting, although other changes perfectly characteristic may occur in the optic papilla. As the discussion tonight is entirely on papilledema, the fundus changes which occur in the conditions just mentioned naturally do not come under discussion. They are mentioned, however, in order to emphasize the vast amount of information which may be obtained from an examination of the fundus oculi in each case.

Until I started to write this paper I do not think that I appreciated what a great advantage it would be to every otologist if he were to familiarize himself with the use of the ophthalmoscope and thus be able to inspect the optic papilla in each case of middle ear suppuration which came under his observation. I would even go further than this and say that it would be of value to examine the fundus in each case presenting aural symptoms which were not entirely due to a nonsuppurative middle ear condition. It is only recently that the true value of an extensive examination of the integrity of the eighth nerve in locating intracranial neoplasms has been fully appreciated. I for one feel that it would be very wise to be familiar enough with the use of the ophthalmoscope to give a fairly intelligent opinion on the condition of the fundus in every case.

The Significance of Papilledema in Neurosurgery. Dr. William Sharpe.

In the diagnostic and surgical treatment of intracranial lesions, a competent ophthalmoscopic examination is possibly the most important single test in a neurological examination; without a careful fundal investigation, no neurological examination is complete. Normal fundi, naturally, do not exclude intracranial surgical lesions.

The term papilledema of the degree of "choked disks" presupposes the earlier milder stages of papilledema of the degree of edematous blurring of the nasal halves and temporal margins indicative of a mild increase of the intracranial pressure and these findings can be confirmed or disproven by the accurate measurement of the pressure of the cerebrospinal fluid at lumbar puncture with the spinal mercurial manometer. Experimentally, the various stages of papilledema can be carefully studied by producing an internal hydrocephalus in dogs. No edema of the optic disks in the absence of sinus disease and local infective processes and in patients who are not myopic should be considered within normal physiological limits without an accurate registration of the pressure of the cerebrospinal fluid, using the spinal mercurial manometer.

The ophthalmoscopic findings in the following conditions were briefly discussed: Tumor, abscess, traumatic hemorrhage and cerebral edema, and in the internal and external types of hydrocephalus.

The following conclusions were expressed: careful ophthalmoscopic examinations are of the greatest importance in the differentiation of many intracranial surgical lesions; the signs of moderate intracranial pressure should be recognized and it should be realized that "choked disks" occur only as the result of high intracranial pressure; the spinal mercurial manometer is the most accurate means of determining the intradural pressure.

DISCUSSION.

DR. DUEL congratulated the readers of the papers on their illuminating presenting of the subject from various angles, and said it would seem superfluous to attempt to *add* anything to the picture. However, he would summarize a few of the points brought out as they seemed to affect the otologist.

In a general way, it appears that the presence of papilledema is, even in its *slightest* form, evidence of either a *considerable* intracranial pressure or possibly a slight inflammatory process involving the optic nerve. In its graver forms it is an evidence of either a *very high* intracranial pressure or a *marked inflammatory process* extending along the optic nerve. Or, apparently in either case, the evidence as presented in the eye may be due to both pressure and inflammatory extension.

It would seem, therefore, that in many instances a marked papilledema might be present without any great increase in intracranial pressure, and, *per contra*, only a slight papilledema might be present in a case in which there was a *very high* intracranial pressure. This is particularly true in cases in which the ocular examination happens to be made early. Inasmuch as prompt action may often mean the saving of vision—on the one hand by the eradication of an infective focus, be it intracranial, in the absence of an abscess of either cerebrum or cerebellum—a septic sinus thrombosis, a labyrinthitis, a localized meningitis, etc., as mentioned by Dr. Dench; or extracranial, in the nasal accessory sinuses, tonsils or teeth, as mentioned by Dr. Faulkner; or, on the other hand, by the removal of the cause of pressure in the shape of any of the intracranial lesions just mentioned, or any of the tumors, or traumatic injuries, mentioned by Dr. Sharpe—it would seem wise in every case where papilledema was present to measure early the intracranial pressure by spinal puncture with a mercury manometer; also to carry out the same procedure in every case of suspected intracranial lesion, even if the eye examination failed to show a papilledema. This plan—early effort to forestall the evil results of long-continued high intracranial pressure by spinal puncture and withdrawal of fluid, or the more certain decompression operation as advised by Dr. Sharpe—would frequently avert a much impaired or even complete loss of vision.

On the other hand, it might save needless repeated spinal taps for withdrawal of fluid, or even a decompression operation in cases where the choked disc is due to infection largely without much increase in intracranial pressure.

In subtentorial lesions, where the equilibrium is upset, the otologist may be of great assistance in a differential diagnosis by elaborate tests of the static sense. It is well for the otologist to bear in mind the point which Dr. Sharpe brought out so forcibly, viz.: that any subtentorial lesion, even a *slight* one, may cause a marked papilledema; while frequently a large lesion above the tentorium might be present without any evidence in the fundus. This is significant, inasmuch as such a large percentage of intracranial abscesses of otitic origin are above the tentorium.

In closing, Dr. Duel reiterated the point that while the presence or absence of a papilledema is of tremendous importance to the otologist dealing with an intracranial lesion, a knowledge of the amount of intracranial pressure, as shown by the manometer, is of even greater importance in cases both with and without papilledema.

DR. PENFIELD said that in the past it has been the custom of the ophthalmologist when confronted by papilledema and failing vision to urge immediate decompression. But the time is past for doing subtemporal decompressions blindly, simply to relieve the papilledema. It is now pretty well accepted that lesions in the midline or in the posterior fossa, which give rise to hydrocephalus, are not benefited—or for a very short time—by decompression. On the other hand, when there are lesions which need elbow room above the tentorium, much benefit may follow and vision may be saved by doing a simple decompression; furthermore, we have means now, with various methods of examination, which should make it almost never necessary to do a subtemporal decompression blindly for an expanding lesion; in addition to neurological examination and X-ray examination, we have air and dye injections and other procedures which make it almost always possible to localize the lesion. The co-operation of the various specialists involved should res-

sult, not in doing a palliative operation, but in the accurate localization and, if possible, eradication of the lesion.

DR. MAYBAUM said that Dr. Dench's remarks about the necessity for otologists becoming better acquainted with the use of the ophthalmoscope were well taken. Three years ago a child, age 8 years, was admitted to the pediatric service at Mt. Sinai Hospital, with a history of inflammatory joints and a septic temperature. There was no history of discharging ears; there was, however, a story of pain in the right ear for twenty-four hours' duration a number of weeks ago. The blood cultures were positive—streptococcus hemolytic. Except for slight thickening of right drum, ear examination was negative. It occurred to someone that it might be well to examine the fundi. A fairly well marked bilateral papilledema was found. On the strength of this finding, with a history of pain in the right ear (no discharge) a few palpable cervical lymph glands and a bacteremia, a diagnosis of primary jugular bulb thrombosis was made. At operation—mastoid sclerosed—no pus in antrum. Lateral sinus contained fluid pus. The child made an uneventful recovery. Since this experience it has been a routine practice in Dr. Friesner's service to examine the fundi in all our ear cases. In four or five cases since the one cited, the eye findings were of considerable aid in arriving at an early diagnosis of primary bulb thrombosis. The important point is to recognize the early eye changes and to note the changes from day to day. We have observed that even after a focus has been dealt with thoroughly, at times the eye changes may continue to advance, despite the fact that the patient is making a satisfactory recovery.

Dr. Maybaum then told of another child admitted to the ear service at Mt. Sinai Hospital, with a history of having had a simple mastoid operation done four weeks before. There was a story of a chill and high temperature for five days. A bright red patch, suggestive of erysipelas, was observed below the affected ear. A blood culture was taken at time of admission and reported positive—streptococcus hemolytic—the following day. By this time the diagnosis of erysipelas was definitely established. The question arose as to whether we were dealing with a bacteremia from a sinus thrombosis or from erysipelas. Most of the men who saw the child were of the opinion that erysipelas does not cause a bacteremia. The ophthalmologist reported definite changes of an optic neuritis. The latter finding, in this instance, did not aid in clearing up the diagnosis. Dr. Maybaum thought it best to temporize for a while. No blood cultures were taken for five days. In the meantime the eye changes were increasing and the temperature was following an intermittent course. All blood cultures after the first positive report were negative. The child had a stormy convalescence from erysipelas, but made a complete recovery without operation. The eye changes in this case were due to toxemia of erysipelas.

Fundus changes should be recognized as very important if present, but should not be relied upon exclusively in reaching a decision for or against operation. The importance of early fundus changes and repeated eye examinations should be impressed upon our minds as aids in diagnosis of otitic complications.

DR. DENCH said that the discussion had been very interesting and very illuminating, particularly Dr. Sharpe's experimental work, although he could not quite agree with all of the statements made, for in almost all of the cases of temporosphenoidal abscess occurring in his own practice there was papilledema. That has been regarded as an important sign. Dr. Dench said he had seen about thirty cases of brain abscess, most of them being temporosphenoidal ones, and in 75 per cent of them there was a papilledema.

Referring to cases of jugular puncture in which the appearance of choked disc occurred rather late, in the first case where the choked disc was called to his attention following operative procedure, the patient was cured, and a little later the ophthalmologist reported that the boy was going blind and that he did not know the reason for it. Dr. Dench pointed out that in operating the intracranial return circulation had been cut off on one side. Lumbar puncture was done, and the vision returned. Papilledema sometimes comes on late, sometimes not at all, and invariably the cases are relieved by lumbar puncture.

It was very gratifying to hear Dr. Faulkner note a total absence of eye symptoms where the accessory sinuses were normal. Presumably there are some cases where there is an accessory sinus disease without gross pathology. It is a question whether such patients would not have gotten well whether operated upon or not. For a long time the rhinologists have been erring in operating upon a large number of cases which showed eye symptoms but with no visible signs of disease of the nasal sinuses. If there is a perfectly normal sinus, it is not clear how there can be any retrobulbar neuritis as the result.

DR. BERENS expressed his interest in the papers and discussions and especially in what Dr. Sharpe had said in regard to the spinal mercurial manometer.

Dr. Berens had always understood that it was dangerous to do a spinal puncture in some patients with papilledema and had therefore suggested the use of Baillart's pressure gauge. With this instrument the presence of increased intracranial pressure can be detected without a spinal puncture, even before any changes can be detected by ophthalmoscopic examination.

DR. SHARPE said he was very glad to hear Dr. Duel speak so encouragingly about the use of the spinal mercurial manometer. A few years ago it was not considered an accurate means of estimating intracranial pressure, but now the consensus of opinion seems to be that it is a very important diagnostic aid in considering subdural lesions. Naturally, if the lesions can be localized and removed it should be done; but if it is a supratentorial one, or only suspected and not localized, then a subtemporal decompression should be performed rather than not. If the lesion is suspected to be a subtentorial one, naturally the operation is through the suboccipital area; however, there are lesions that can be diagnosed and localized and not removed, such as tuberculomata and infiltrating gliomata. In that type of case a decompression operation should be performed, subtemporal or subtentorial, as indicated.

As to localizing the lesion by air injection, there is still a large field for investigation. In the last three brain tumors observed by him, sarcoma, glioma and a tuberculoma, the air injection of the ventricle did not help in the localization, neither did it give any help in a case of another circumscribed glioma or the ventricles show any malformation whatever. So, rather than permit a patient with increased pressure to go on to impaired vision because the lesion cannot be localized, a decompression operation has a definite value.

Dr. Sharpe said he was interested in hearing about the instrument Dr. Berens mentioned and would like to know more about it.

SECTION ON LARYNGOLOGY AND RHINOLOGY.

February 24, 1926.

I. Reading of the Minutes.

II. Presentation of Cases.

III. Papers of the Evening.

a. The Application of Bacterial Vaccines Locally in the Treatment of Nasal Sinus Conditions. Dr. Harold Hays.

Discussion by Dr. Thos. S. Winslow (by invitation), Dr. Arthur Palmer.

b. Lantern Slide Demonstration of the Anatomy of the Accessory Sinuses of the Nose. Dr. Simon L. Ruskin (by invitation).

IV. General Discussion.

IV. Executive Session.

The Local Administration of Bacterial Vaccines in the Treatment of Subacute and Chronic Nasal Sinus Conditions. Dr. Harold Hays.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. WALTER M. KRAUS said that great credit should be given Dr. Hays for the pioneer work that he has done. He related that when abroad he happened to learn that Professor Besredka, of the Pasteur Institute of Paris, had been working on the production of immunity by a method quite different from that usually accepted, namely, immunization by mouth.

Dr. Kraus quoted the following from an article on the subject in the *Lancet* in 1922:

"An attempt had been made to infect mice with B. Paratyphosus B by the mouth, but the results had been entirely negative, even when massive doses of the living organisms were given. At the same time further experiments were going forward in his laboratory in which control animals were inoculated subcutaneously with fatal doses of this organism in order that the degree of resistance of other mice, which had been immunized in various ways, might be compared with that of the normal unprotected animal. Being on one occasion short of mice, Besredka used as a control one of the animals which had been subjected to the unsuccessful attempt at mouth infection just mentioned. To his surprise he found that the animal remained none the worse for an injection of paratyphoid organisms, which was considered surely lethal. On pursuing the matter further, this resistance was found to be present in varying degrees in all of the other animals which had received the meal of organisms. The investigator at once realized that he had stumbled across a new fact of great importance, and set himself systematically to study the possibilities of producing immunity by the ingestion of organisms in the case of those diseases where the infecting agent enters the body by the alimentary canal."

You may be interested to know that an inoculation against dysentery is not possible by the hypodermic method on account of the extremely toxic results, but by mouth it has become possible. Experiments on large control groups with typhoid and paratyphoid vaccines given by mouth were more successful in conferring immunity than the same given by injection.

I know of a man injured during the war who had a wound which was still discharging. The Besredka vaccine was applied to this large discharging sinus in his thigh, which had resisted all treatment; the sinus healed up in three weeks, and the man had no further trouble.

The immunity conferred by the Besredka method is produced within the cells of the organ which has a specific affinity for that particular germ—for instance, the intestinal mucosa for the typhoid bacillus.

It is interestng in this connection that Dr. Flexner in his classical work on epidemic meningococcus found that the best way to give the serum is in the spinal canal. The treatment of tetanus illustrates the same thing. The serum is given intraspinously and intravenously, and sometimes intramuscularly, but the most efficient way is intraspinously, so there again you see that the vaccine is brought in closest contiguity to the structure which is affected by the organism.

There are various other ways in which this same immunity may be conferred; Dr. Hays has mentioned them—in gynecology, in skin diseases and in general surgery.

The reason that the immunity when produced by hypodermic injection takes time to develop, is that it takes time for the dead bacilli to disintegrate and reach the cells in which the immunity is to be produced. When given directly—as typhoid by mouth—the immunity naturally results more rapidly.

In closing, it would seem from reading some of the literature that the greatest result in general that has been accomplished up to now has been with typhoid, bacillary dysentery and streptococcal infections.

DR. T. S. WINSLOW said that he was in a position to hear the different views of various members of the profession on the different types of vaccination, and it was most interesting to compare them. One hears of miraculous results from one doctor or another, but they are few and far between. The whole question seems only a shot in the dark as yet; though the idea presented by Dr. Hays seems to have much promise if more experimented on.

There are one or two points I would like to mention tonight. If we let the bacteria grow in broth in the incubator for four days, generally at the end of

that time there are no living bacteria in the culture. Why not use this direct without sterilizing it by heat? In this way we might get the benefit of any bacteriophage phenomena that might be present. Or, why not take the Berkerfelt filtrate from the bacterial growth and use that direct? It would contain all the endotoxins in their unaltered form. Of course, I do not as yet suggest giving these by hypo. I would like to hear these suggestions discussed.

DR. ARTHUR PALMER said that during the past two years he had had the opportunity of seeing a good many of these cases which had been treated by Dr. Hays with Dr. Winslow's autogenous vaccine applied locally to the nares. At first he had been inclined to be skeptical of its value. Many questions came to mind. In the first place, many could understand how an injected vaccine would reach the vascular system and create antibodies which would react unfavorably to an infection. Many had tried that, and sometimes considered the results good, though there were many disappointments; but how could a vaccine applied locally to a moucous membrane affect the organisms? Could it be absorbed and affect them as it did when injected, or did it have a merely local effect—bacteriocidal locally or mechanically—whereby through increase of mucus the organisms were washed out, or was it ingested and absorbed through the intestine, where the absorption is much more rapid than in the nares, and thereby affected unfavorably the infection? Some of these questions had not yet been convincingly anwered, but he had been won over to the use of vaccines locally for two reasons; first, by the testimony of the patients, many of whom had returned without solicitation and told of the excellent results from the vaccine treatment. Second, many of the patients have been observed over a considerable period of time and had shown improvement in the nasal and throat conditions. The end of the rope has not yet been reached, but the matter is still under investigation.

Anyone can understand the ease of using the vaccine locally as against injecting it. In the first place, the patients object to the needle, whereas they very rarely object to the tamponing of the nose with the vaccine; moreover, the local vaccine can be easily used at home by the patients, who can spray the nose two or three times a day; and they have no unpleasant reactions such as they get from the hypodermic injection.

As an illustration of how science has advanced in the same way children learn as they grow up, i. e., by observation and investigation, Dr. Palmer told of his little son, less than two years old, who while sitting on his mother's lap looking over the current periodicals saw the picture of a clock and said "tick tock," put his head down and at first seemed to think he was going to hear it, but then looked up at the real clock and listened. He will certainly soon learn to distinguish between the false and the real. Dr. Palmer said he was sure all would be interested in observing and investigating this new method of treatment, and that when all was said and done would feel that Dr. Hays had made a valuable contribution to our armamentarium in the treatment of conditions which often heretofore had baffled our best efforts.

DR. FREUDENTHAL said that all those who had had any experience with nasal diseases have come across a good many cases which are extremely stubborn and nasty, and difficult to deal with. All have seen cases that have been operated upon over and over again, here and elsewhere, and have returned after maybe ten years of treatment with a profuse discharge still present. In view of this, he was glad to welcome anything that promises good results. What Dr. Kraus has said about immunity was most interesting.

Dr. Freudenthal said he was convinced that he had himself acquired immunity against tuberculosis. He had been a very delicate youngster, and was put in charge of one of the hospitals where he was called upon to treat tuberculous laryngitis cases, and had been doing such work now for thirty-one years. Everyone knows how such patients act, coughing and spitting at you. But after working with them for thirty-one years he is still alive and well, and could regard himself as illustrating the theory of acquired immunity as brought out by Dr. Kraus.

Dr. Hays had stated that the vaccine was applied with gauze; was that simply a local application, not a spray?

Dr. Freudenthal further said that he had not had any good results from hypodermic injections of these vaccines, but was a great believer in local treatments. A few years ago he had read a paper before the Triological Society describing a new form of application—bougies of boric acid or ichthyol put up in the nose and left there for some time to produce a direct local effect. This new method of Dr. Hays' seemed very promising, and he hoped to hear more about it.

Dr. Seymour Oppenheimer said that he had had no experience with this method of treatment, but that at the Willard Parker Hospital in some of the services they use antitoxin applied locally as well as hypodermatically. He was not prepared to give figures as to the relative mortality of those treated locally as against those not so treated, but is convinced that the nasal condition does clear up much more rapidly, and the membrane in the nose disappears more rapidly, and the disease is much more rapidly eliminated than in those not treated with the local applications.

Dr. Welch said he understood that the treatment was especially applicable to chronic conditions, and would like to know from Dr. Hays whether polypi disappear in the course of the treatment. Also he would like Dr. Hays to compare this treatment with the argyrol tampon treatment described in one of the papers read before the Section last year.

Dr. Hays stated that he had been much encouraged by the discussion on the paper. When the subject was presented at the meeting of the American Medical Association last year, either because it was not particularly interesting, or for lack of time, or because no one knew anything about the subject, it received little attention. He hoped that the members would try the treatment, in order to give their reports on it. Apparently there is quite a difference in the preparation of the vaccine. When Dr. Winslow was away from the laboratory, someone else made up the vaccine and the results from it were very unsatisfactory.

The subject is a very interesting one, not only in sinus conditions, but in various types of infection, including bone infections. Dr. Besredka spoke of various types of osteomyelitis that had been cured by these vaccines, persistent acne, etc. Everyone is familiar with cases of delayed healing of mastoid wounds. In a number of these cases he has had cultures prepared and dropped the vaccine into the wounds, and many of them have healed up promptly. An attempt had been made in treating tonsil infections with the vaccine, but so far with little success. Dr. Winslow had suggested using it by injection behind the tonsils. Thus far we have used it by immersing strips of gauze in the vaccine and packing it in the nose as far back as possible, and having the patients use the vaccine at home in the form of a spray or with a medicine dropper. The vaccine has also been used in surgical cases. One of the hospital nurses had an infection of the finger that could not be cured by ordinary surgery, but it healed very nicely when the vaccine was used as a wet dressing. One of the gynecologists is trying it in lesions of the cervix, where it can be applied easily and the results are waited with interest.

Answering Dr. Welch, Dr. Hays said that long before the matter was brought up at the Academy last year, he had been using the Dowling tampon, but he considers argyrol a messy thing to use, and it is questionable whether it does any more good than to stimulate the mucus secretions.

Dr. E. M. Pullen said that he had been using the vaccines on some cases of atrophic rhinitis that had previously been treated with various other agents, the patients varying in age from 12 to 56 years. The treatment was not continued with all of them, however, for they would not follow out instructions. His procedure was to first irrigate the nose thoroughly with an alkaline solution, and carefully remove any adhering particles, etc. Then small pledgets, half an inch long and very thin, were saturated in the vaccine, and pushed up against the posterior wall and left in place for an hour. Then the patient is given a spray of the solution to be used at home two or three times a day; and in every case so treated the mucus and discharge is greatly lessened at the end of three weeks, etc.; and then the odor diminishes, etc. One patient, a woman, who had suffered from the condition since she was a child, was entirely cured. You cannot put back the mucous membrane, but the mucopurulent condition can be cured.

SECTION ON OTOLOGY.

March 12, 1926.

Presentation of Lantern Slides of the Mastoid. Dr. George S. Dixon.

DISCUSSION.

DR. DIXON showed a number of lantern slides of prepared bones of various types, and skiagraphs of similar bones in the living subject.

DR. LAW declared that he could only say that Dr. Dixon had given a very beautiful pictorial representation of radiographs of the mastoid structures. He had been especially interested in what Dr. Dixon said about radiographing a child's mastoid. There are few things so annoying as to have a request come from the children's ward and to have to radiograph an obstreperous baby; it is a very difficult thing to do, and the radiogram does not give much information. The plate Dr. Dixon showed was a very excellent one. All one can expect to get is the anatomy; there is not much mastoid there as a rule; and it is practically almost useless to attempt to get the radiograph of an infant's mastoid except under an anesthetic. Dr. Dixon's pictures of the adult mastoid were very enlightening, particularly in that they showed a group of the main factors that can be pointed out on an X-ray plate and the meanings of the plate interpreted.

DR. DIXON said that he could add nothing excepting that as a rule it is almost useless to attempt to obtain the skiagraph of a baby's mastoid, under 2 years of age. He had seen one girl, age 5 years—and occasionally younger—who had a perfectly beautiful, well developed pneumatic mastoid, but as a rule one cannot hope to get much under four years. One reason for that is that it is difficult to handle the children until they have reached the age of reason, and often next to impossible to do anything with them excepting under an anesthetic; when they are under two years of age one can usually depend on finding the sinus well forward, and you may find some pneumatic cells; you can always count on finding an infantile bone in a child age 2 years or under. It has been his practice to rather discourage the taking of skiagraphs of children under 2 years; but it is possible to get good results beginning about 4. If you put a child under an anesthetic, unless you have a very rapid machine and can expose them with a flash, you are not likely to get very good results; but if you can win the confidence of the child, and can make it understand what is needed, good results may be obtained. In one instance an excellent skiagraph was obtained of a child, age 21 months, with a large mastoid dressing on each side. He rather surprised himself, but happened to get the child perfectly quiet and got one picture, and then got it quiet again and took the other. The child was running a high temperature, and it was important to know where the sinus was and whether any cells had been missed. Under an anesthetic the results are unsatisfactory, for the child goes under crying and sobbing and tosses the head from side to side; and if they are deeply anesthetized you still have to bind the head down to be reasonably sure of good films.

DR. HELLER said he was very glad indeed that Dr. Dixon and Dr. Law had spoken as they did about the skiagraphs of children under 2 years. He then cited an instance of a very excellent otologist who had been severely blamed for not having had an X-ray picture of a child, age 3 years, who went on to a mastoid operation. The family claimed that he had been negligent and had the X-ray been taken, the child might have been saved the operation. The otologists and the X-ray men ought to co-operate in spreading the information that excepting under special conditions the X-ray picture of a child age 4 years is worth very little.

Abstract: **X-ray Examination as an Aid to Mastoid Surgery.** Dr. Frederick Whiting.

This paper is devoted largely to a consideration of the various types of mastoid structure which can be recognized by the ordinary flat plate. It also gives exhaustive consideration to those anomalies of mastoid structure which can only be appreciated by properly projected stereoscopic X-ray plates.

The stereoscopic plate demonstrates the arrangement and situation of deeply placed nests of cells which escape detection where dependence is placed

upon the flat plate alone. The existence of these cells is often responsible for collections of pus which remain undrained after the ordinary mastoid operation because their presence is not recognized before operation. The stereoscopic plate would have furnished the operator a definite knowledge of the existence of these cells and probably would have saved the patient from a secondary operation.

DISCUSSION.

DR. DIXON referred to an occurrence some years ago when Dr. Whiting had read a paper on resection of the internal jugular vein. The late Dr. Gruening arose and said: "Mr. Chairman, who shall speak after the King?" He felt somewhat that way himself tonight. Referring to what had been said about sclerosed bones, he called attention to one reason why a brain abscess, for instance, may follow an infection of that type of bone. He had seen a large number of bones of that particular type with a dehiscence in both the tegmen antri and tegmen tympani, and of course the process would extend in the line of least resistance.

Dr. Whiting had emphasized the importance of stereographs. There is no question but that good stereographs of the mastoid are very useful, but it does not seem necessary as a routine measure. In selected cases it is not only advisable, but is often imperative. That, however, had been covered by what was said about very obscure cases.

The X-ray men have urged the importance of co-operation on the part of the surgeons; in a large number of cases it is hardly worth while to simply send films to the surgeon with a report. The surgeon and the X-ray man, as Dr. Law has often urged, should get together and discuss the plates in connection with the history of the case. There are a few surgeons who consult regularly with their radiologists and from them has been obtained more information than from probably three-fourths of the other work done.

The question of capsulatus infection had been discussed before the Section many times, and it was hardly worth while to go over it again, except possibly to cite one typical case. Two or three years before the death of the late Dr. Adams he was consulted by the director of a large institution for an ear condition. He was quite deaf, but there was no pain, no tenderness, no temperature, and decreasing discharge. The question was whether it was best to operate or to wait. Dr. Adams and Dr. Lewis looked over the case. The patient looked better than he did before and said he felt and could hear better, and he wanted to know whether it was possible to wait another twenty-four hours. It was decided that he could. The next day at noon Dr. Adams operated and found the dura exposed for about three-eighths of an inch.

Dr. Dixon said he did not wish to appear as seeking more work for the X-ray men, but called attention to the fact that if those persons who have any tendency to ear trouble whatever would have their mastoids X-rayed while in good health and the films filed, they would be invaluable in case of later infection.

DR. LAW said there was no possibility of adding anything to such a wonderful presentation of such a difficult subject. Not a point made could be objected to, and nothing had been omitted. It only remained to congratulate Dr. Whiting on his very fair and level-headed presentation of the value of the X-ray in mastoid surgery. However, one or two points might be emphasized, one especially. Dr. Whiting had spoken of good plates. Dr. Law said he had been called upon to pass judgment on plates where it was difficult to say whether it was a picture of a mastoid or of a frontal sinus. He will not accept a plate for interpretation that does not show the texture of the surrounding bone—not the diseased part only, but the surrounding bone.

So far as stereoscopy is concerned, Dr. Law said he fully agreed with Dr. Whiting. It is employed universally in private work, but not in rapid work in the clinic. Fortunately, there we can get the patients back again if there is uncertainty. The one unfortunate thing about the stereoscope is that many surgeons cannot use it. Another point to be emphasized was what Dr. Whiting said about consultations. Dr. Law said that for years he has tried to secure consultations between the Roentgenologist and the surgeon. A few men do not recognize the value of this and study the plates together,

but the vast majority of the surgeons accept the statements on paper, and some of those interpretations he has had to have interpreted to him. If the surgeons would come to the X-ray rooms and get acquainted with the Roentgenologists and learn to use the stereoscope, they would learn to appreciate its advantages, particularly in a pneumatic type of mastoid. Where the clinical evidence is vague it is important to know whether or not to operate. With a single plate one is liable to see cell structure and conclude the disease has not progressed far. In the stereoscope one will see the same structure, but it will prove to be the cells under the cortex, and the cells down deep between the sinus and the posterior wall to be softened and broken down. That will not be shown by the single plate, but can be shown by the stereoscope.

Sometimes the plate will convey the wrong impression. In one case a nurse had a very severe capsulatus infection. The plates revealed quite a cavity, as large as a thumb, but she refused operation and eventually recovered. Three months later she came to the hospital and another plate was made; there was a pneumatic mastoid, the cells all cleared up, but right in the center was a punched-out area as large as a thumb. Three years later she came in again, and another plate was made, and she had a reformed mastoid. Supposing at the end of three months she had been somewhere else and had developed a mastoid with mild symptoms; an X-ray picture would have been made and they would have found this hole; this absorbed area would have been attributed to present process; it might not have been of long enough duration to warrant the destruction that the radiograph showed. It makes no difference whether the absorption was caused by the present or a previous attack; the damages had been done, and the second attack started in just where the previous attack stopped. That was an instance in which the X-ray would seem to indicate more trouble than the clinical evidence would warrant. It cannot be too often urged that the surgeons and the Roentgenologists should get better acquainted with each other and study the cases together.

DR. GOALWIN: Dr. Whiting mentioned some of the interesting theories in regard to the causes of the undeveloped mastoid, but said nothing about the causes of the overdeveloped mastoid. Hyperpituitarism is one of the most frequent causes of overpneumatization of the mastoids. It is associated with overdevelopment of the paranasal sinuses, and is one of the earliest signs of acromegaly.

The compensatory type of overpneumatization of the skull, which has been described by Schüller, should also be mentioned. In these cases the skull becomes highly pneumatic in an effort, so to speak, to fill up the vacant portions of the cranial cavity after an arrested hydrocephalus. This condition may be associated with a compensatory concentric hyperostosis. Clinically, it is important to keep this in mind because the shadow of the dense bone will be superimposed on the mastoid cells and produce an apparent clouding. Since hyperostosis is also frequent in acromegaly the above statement applies also to these cases.

It should also be remembered that tumors of the mastoid are not at all infrequent. One who attempts to diagnose disease conditions of the mastoid from the Roentgenogram should therefore be prepared to make a differential diagnosis between tumor and inflammation. There are, furthermore, certain typical changes in the mastoid and petrous pyramid, which are produced by tuberculosis. These should be looked for in all chronic cases.

Although my experience has not been as extensive as that of the speakers of the evening, I am sorry to have to disagree with them in regard to the value of the X-ray examination of the mastoids in infants and very young children. About half a year ago I made such an examination on an infant, age 7 months, and to my great surprise found perfectly developed pneumatic mastoids. On the affected side there was definite evidence of bone softening, which upon operation proved to be caused by the streptococcus hemolyticus. Another patient, age 25 months, also had a well developed mastoid, in which the pathology was easily demonstrated Roentgenographically and proved by operation (Dr. MacKenty).

The Bucky diaphragm enables us to bring out the very small cells and the very fine septa in the mastoids of these infants and young children.

The examination is, of course, difficult and time consuming. If one wishes to save time, it is easy to instruct the parents or nurse to give the child 33 to 5 grains of chloral hydrate by rectum, or half that amount by mouth. I prescribe an aqueous solution containing 1 grain per c.c. If this is given about one-half hour before the examination the child is sufficiently drowsy and the examination can be made without difficulty.

The routine X-ray examination of the mastoids in infants and very young children should not only help us in diagnosis of disease but should enable us to tell how early the pneumatization of the mastoid can take place.

SECTION ON LARYNGOLOGY AND RHINOLOGY.

March 23, 1926.

An Unusual Case of Rib Transplant. Dr. Francis W. White.

W. D. About seven years ago, while in the army, a septal abscess developed in this patient, which was opened and drained. This was followed by a very large perforation of the septum and the falling in of the bridge of the nose. A year ago last January, under general anesthesia, a portion of a rib was removed and transplanted in the nose by the intranasal route as advocated by Corti. The result was satisfactory. A few weeks after all appearances of reaction had disappeared, a slight shifting of the transplant to the left began. This involved the upper end of the graft more than the lower. Finally, after several months of very slow movement to the left, it became stationary; the upper end of the graft being in proximity to the inner angle of the left eye. The bulging of the lower end of the graft, covered with mucous membrane was discernible in the left nasal chamber. The graft became fixed in this position.

About five weeks ago, under local anesthesia, and by the intranasal route, the graft was freed, as were all the tissues of the bridge of the nose and downward on each side toward the cheek. In order to free the graft, however, it was necessary to chisel it away from the nasal bone of the left side. Due to the inordinate amount of force required to drive the chisel through this bony connection, it was decided to try actual fracturing by means of heavy forceps. This was accomplished. The graft was then quickly removed and about one-quarter of an inch was removed by means of heavy scissors; reintroduced through the original incision; put in place; and by means of a retention apparatus was held therefor about ten days.

Interesting features of the case are, first, the entire absence of bony union between the upper end of the graft and the frontal bone; second, the exceedingly firm attachment of the middle portion of the graft to the nasal bone; third, the fact that the graft was removed, shortened and replaced, the appearance of the graft being that of a piece of bone having been removed for the first time; fourth, the cause of the wandering of the graft was, first, unequal contractions of the tissues about the graft and, second, the length of the graft possible having been too long.

DISCUSSION.

Dr. W. W. Carter said that the reason the graft wandered was that it was not anchored, as it should have been at the time of operation, to the frontal bone under the periosteum over the nasofrontal process. You get your osteogenesis from the frontal bone itself. The so-called periosteum over the frontal bone is a misnomer, as this is not a true periosteum, the frontal bone having been developed in the membrane; the periosteum, or rather aponeurosis, merely serves to retain the transplant in position, and the frontal bone having been excoriated by the chisel and the graft mortised in, you get your osteogenesis from this bone. It is very interesting to know that in this case, even though not anchored to the bone, there was no absorption of bone. This, no doubt, was due to the fact, as Dr. White himself said, that the bone was in

contact with the frontal bone and there was an exchange of osteogenesis between the transplant and the nasal bone which had been excoriated.

Dr. Carter said that on two occasions when the transplant had slipped, he had removed it in order to put it into a better position. This will occur occasionally, due to cicatricial contraction which may pull it away from the frontal bone. It is always difficult to remove the graft, for the transplant forms a very firm union with surrounding tissue. Such cases as this one of Dr. White's confirm Dr. Carter's original contention, made many years ago, that an autogenous bone transplant, properly introduced into the tissues of the nose and contacted with live skeletal bone, remains alive and grows, its growth being regulated by the functional demands of the part.

Dr. White agreed with Dr. Carter about mortising the graft in the frontal bone, but insisted that the graft was probably a trifle too long so that the pressure that ensued at the lower end was greater than the resistance at the upper end, causing it to slip out.

A Case of Developmental Deformity of the Nose Corrected by Bone and Cartilage Transplants. Dr. W. W. Carter.

(*Published in full in this issue.*)

Closure of Bucco Antral Fistula. Dr. Irving Wilson Voorhees.

This private patient was shown at the January meeting of the Section. For several years the gentleman had suffered with pain in the right superior maxilla. Most of the teeth had been drawn and an attempt had been made to do an antrum operation through the alveolus, which was unsuccessful. Under ether at the Manhattan, I removed a bony growth, which is probably an odontoma. The soft tissues would not heal and the resulting fistula was about an inch long and three-eighths inch wide.

The patient was presented in January with the purpose of getting some suggestions from members present regarding closure, but none of these seemed quite practical because of the difficulty in carrying them out.

On March 1, under ether, at the Manhattan a large plastic flap was dissected free from the inside of the cheek, the incision extending well up into the antrum. The mucoperiosteum covering the hard palate was dissected free over the entire right half. Three mattress sutures were introduced from the midline and inserted deep into the cheek flap, thus serving as retention sutures. This done, the freshened edges were united by interrupted chromic sutures and the patient was returned to his room. The mouth and nose were kept clean with simple washes, and mercurochrome, 220, 5 per cent, was applied on both surfaces of the suture line several times daily. Recovery was uneventful and the union was so perfect that it is difficult to find the lines of incision.

Presentation of Instrument. Dr. Joseph H. Abraham.

Dr. Abraham presented a modification of his antrum needle, devised twenty years ago. It has been provided with a universal attachment so that it may be used with the Luer syringe instead of the one that originally came with it.

The Present Status of Plastic Surgery in France. Dr. Leon Dufourmentel. (Abstract by Dr. J. Eastman Sheehan.)

A lecture in English, even on a familiar subject, is for me an unusual experience, but the welcome I have received in the past month encourages me to hope you will excuse my errors. I came to the United Stats on an official mission from my government, to inquire into the position of plastic surgery here. Incidentally, as on this evening, I have endeavored to make known its status in France.

There are only a few of us in France who practice this surgery; as one of those who do so, I have been chosen for this mission. After Nelaton and Ombredanne, who made it a part of their work, the first with us to master the subject were Morestin and Sebileau. These conducted, in Paris during the war, the most important services involving the application of plastic surgery to war wounds. Outside of Paris, the chiefs of hospitals devoted to this work were pupils of Sebileau, among them Lemaitre at Vichy, and my-

self at Chalons-sur-Marne. After the war, plastic surgery was adapted to meet the civil needs and to deal with deformities arising in peace conditions.

At present, there are two courses in which this specialty is taught: one at the Clinic of the Faculté de Medicin, in French, given by Professor Sebileau and myself; the other, in English, at the International Clinic, directed by Drs. Lemaitre, Gillies, Eastman Sheehan and Ferris Smith.

Grafting is the operation by which plastic surgery is distinguished. Skin, bone, cartilage, fat, etc., can be transplanted by now well established methods. For bone, we use the method of Delaquiere, as well as that of Albee. Three special methods of skin transfer deserve special mention: *1.* migration from a distant area, the farther end being raised and reinserted higher up until the area of application is reached; *2.* bipedicle flaps, the skin being left attached at both ends while the flap between is applied; *3.* whole-thickness grafts, first devised by Hamilton in America, generalized by Davis and others in America and by Sebileau in France.

Inseparable from transplant procedures are care in stretching the skin and in the avoidance of scars. Scar avoidance is essentially preventive and is partly accomplished through hidden incisions, as on the hairy areas and the wrinkles and natural cavities of the face. In visible areas, it is only by the fine quality of the suture that the scar can be rendered less noticeable. A final resource is found in tattooing, by means of which over-colored lines can be blanched and over-white hues can be colored. The materials are mentioned in an article I published in the *Revue Medicale*, Dec. 10, 1919.

Skin distension can be safely accomplished by the insertion of bone, cartilage, or fat, where a depression has to be filled. Paraffin must never be used, as paraffinoma is a frequent and very serious sequel. In other instances, as in operation for wrinkles, resection and suturing will restore the normal tension.

In reconstructive surgery, the first care is to respect the natural processes of healing; often, this alone gives an unexpected and indeed wonderful result. Many large mutilations have been repaired by guiding the natural healing (photographs). If the result is in adequate, it can be supplemented by surgical intervention. Lantern slides followed, showing:

1. Grafting half an upper lip.
2. Destruction of a lip by dog bite and repair by pedicle flap, followed by tattoo. Function restored.
3. Bipedicle flap from neck for repair of lip.
4. Same for large orbitofrontal loss.
5. Correction of various nasal deformities; *a.* saddle nose corrected by insertion of cartilage through eyebrow incision or intranasally. In obtaining the cartilage, use a *small* incision in the thorax and separate the muscle bundles horizontally; do not cut across them.

b. Humped and broad nose. Method according to conditions—either excision along the bridge, or release at the base and press together the parts thus separated.

c. Deformities of cartilaginous bridges, hook nose, long nose, pug nose. Correction by suitable excisions from the cartilage and suturing in the new relations.

d. Morestini's operation for rhinophyma by a "modeling abrasion."

6. Correction of abnormalities in temporomandibular articulation.

a. Ankylosis. Bone resection by burr on the normal site of the joint. Curved and narrow as possible. Skin around then closed. Permanent mobilization, to prevent resurrence, is obtained by apparatus of Darcissac—two dental processes, from each of which a long hook passes out of the mouth. The hooks are then connected by a rubber band, stout enough to hold the mouth open; against which is the effort of the patient to close the mouth. Congenital recession of the lower jaw is corrected by cutting the epiphysis and grafting a piece of bone.

b. Luxation: remedied by resection of the meniscus.

c. Cracking of the joint: same treatment.

d. Obliquity and protrusion of jaws: remedied by abrasion of the condyles on the side opposite the obliquity, and on both sides for protrusion, using burr or small biting forceps.

7. Reconstruction of the total loss of lower jaw: Double pedicle flap from scalp, which produces beard. Lip border by mucous membrane flap from upper lip. Jaw replaced by piece of shin bone. Cinema shows this repair in all stages and illustrates restoration of function of lips, tongue, jaw, etc., in an area where destruction was total (patient a German soldier).

8. Distended and elongated breast: Nipple moved to a higher position; surplus tissues eliminated; bulk notably reduced.

DISCUSSION.

DR. JACQUES MALINIAK: It is very difficult to discuss in five minutes a paper which includes all the plastic surgery of the head, a general review of all maxillofacial surgery. The results which Dr. Dufourmentel has shown on the screen speak for themselves. Those horrible facial mutilations which we had to treat during the war in very large numbers have been reconstructed as the pictures show, by one who is both a surgeon and an artist, and with perfect ingenuity. Let us hope, however, that we shall not again have to do with this war surgery.

I shall try to bring out in this discussion some facts that interest us especially in facial surgery in times of peace. Dr. Dufourmentel has presented to us excellent results obtained in surgery of the temporomandibular articulation. Having worked about fourteen years ago with Dr. Dufourmentel in the service of Dr. Sebileau, of Paris, I remember his first attempts in this line. The operation for temporo-maxillary ankylosis and luxation which he attempted at that time seemed difficult and bold, since he approached the articulation by a route which seemed rather dangerous; but the instrumentation, operative technique, and after-treatment by early mobilization were carefully worked out. The results, which I have observed on many occasions in Paris, were very satisfactory.

In regard to rhinoplasty, I wish to emphasize some facts which seem to me important. I agree with the speaker that the endonasal route is the only one to employ in corrective rhinoplasty. There are some rare exceptions to this rule, as in the case of chronic infection of the nasal fossae, which is difficult to control. In similar cases, the incision should be made in the eyebrow, where it remains entirely hidden. As we know, this route was first used by Dr. Dufourmentel. We do not believe that we are justified in mutilating the face with visible scars in order to correct nasal deformities. As to the transplants to be employed in rhinoplasty, I agree with the speaker that cartilage is the substance of choice. Its superiority has been demonstrated by many writers, experimentally and clinically. Yet, in certain cases the patients refuse to give their own cartilage and we have no choice but to use the best substitute. Of all heteroplastic and alloplastic substances, ivory is the best to use. Years of experience have demonstrated this. Its tolerance in human tissues is explained by its structure, which is similar to that of bone. It differs only by absence of water in the ivory and a quantitative difference of magnesium sulphate and calcium. In a paper on this subject, published last year, I brought out the biology and qualifications of ivory. However, whenever it is possible, cartilage taken from the patient himself should be preferred for the nasal transplant. It has also been shown that a free bone graft used for the reconstruction of the nasal dorsum undergoes more or less rapidly a total necrobiosis; but, on account of the strong osteogenetic reaction, it favors reconstitution of the bony segment for which it is substituted, if placed in contact with the bony extremities. These conditions are not present in nasal reconstruction. An intermediate procedure was conceived, in using a mixed transplant of bone and cartilage. I have never had occasion to employ this method nor to observe the results from it.

BOOK REVIEWS.

Plastic Surgery of the Nose. By J. Eastman Sheehan, M. D., F. A. C. S., Associate Surgeon and Lecturer to the International Clinic of Otolaryngology and Facio-Maxillary Surgery, Paris; Plastic Surgeon and Chief of Nose and Throat Clinic, New York Post-Graduate Medical School and Hospital; Member, American Association of Plastic and Oral Surgeons, etc. New York: Paul B. Hoeber, 1925. Price, $10.00.

This monograph of 250 pages crystallizes the highly specialized work of the author. The subject is practically presented, beautifully illustrated and printed and is a real credit to both author and publisher. M. A. G.

Diseases of the Nose, Throat and Ear. Edited by A. Logan Turner, M. D., F. R. C. S., with the collaboration of J. S. Fraser, M. B., F. R. C. S.; W. T. Gardiner, M. C., M. B., F. R. C. S.; J. D. Lithgow, M. B., F. R. C. S.; G. Ewart Martin, M. B.; F. R. C. S.; Douglas Guthrie, M. D., F. R. C. S. With 222 illustrations in the text and twelve plates, 8 of which are in colors. Bristol: John Wright and Sons, Ltd., 1924.

Even a belated review of this excellent volume originally prepared by the late Dr. W. G. Porter will serve to acknowledge the thanks of the Otolaryngologic reader to its practical author. The late Dr. Porter's book on Diseases of the Nose, Throat and Ear, is full of common sense, practical suggestions and lucid presentation; no wonder, then, that three editions were soon exhausted and that it bore the stamp of popularity long before the revision here considered was undertaken.

It is a fine tribute to the memory of Major W. G. Porter that Dr. A. Logan Turner and his collaborators prepared a fourth edition of this book, preserving the underlying features of the original edition and providing the senior students of medicine and the practitioner with a volume of moderate size and containing in concise form, well presented and excellently illustrated, the most necessary subject-matter in practical otolaryngology. M. A. G.

Handbuch der Hals- Nasen- Ohren- Heilkunde by A. Denker and O. Kahler, 1926. Julius Springer, Berlin, J. F. Bergmann, Munchen.

This formidable new treatise has just been issued and contains the otological subject-matter. It is of very unusual importance for its presentation of the comparative anatomy, embryology, laboratory technique and oto-histology, minute consideration of the cochlea and vestibular nerves and post-mortem technique in the examination of the organ of hearing.

The chapter on Phylogenesis and Operative Anatomy, by Wilhelm Brock, Erlangen, brings the human ear in its evolutionary relation quite up-to-date, and is beautifully presented and illustrated. Embryology, Anthropology and variations are considered in Chapter II by Alexander, Vienna, and in the hands of this master is as complete a presentation as that found in the recently written textbook by Alexander and Marburg on Oto-Neurologie. Then follow chapters on Deformities of the Ear by Marx, Muenster, and Applied Anatomy by Körner, Rostock. The fifth chapter by Eckert-Mobius, Halle on Microscopic Technique and Histology of the Organ of Hearing is a feature of this volume, for it contains microscopic technique in every detail and an excellent series of illustrations in oto-histology and orientation methods. Active research workers in labyrinth investigations will find a very acceptable chapter by Anton, Hale, on the Centers and Tracts of the Cochlea and Vestibulary Nerves.

The second section of this volume is devoted to the physiology of the ear and here also must be noted the progressive status in the subject-matter. The chapters on Physiology of the External and Middle Ear and of the Cochlea have been prepared by Schaefer, Berlin, and the succeeding chapters on the Physiology of the Semi-Circular Canals and the Physiology of the Centers and Tracts of the Vestibulary Apparatus are presented by Neumann, Vienna.

The third section includes all phases of Pathology and Therapy and contains the following chapters: Etiology; Affections of the Cochlea and Vestibular Apparatus in Relation to Diseases of the Brain and Peripheral Nervous System, by Beck, Vienna; Neurasthenia and Otological Reflexes, by Ernst Urbantschitsch, Vienna; Endemic Kretenism in Relation to the Ear, and The Ear in Relation to General Bone Diseases, by Nager, Zürich; The Ear and the General System, by Lederer, Dresden; Acute Infectious Diseases, by Stenger, Königsberg; General Pathology of Syphilis of the Ear, by Beck, Vienna; Osteomyelitis, by Müller, Erlang; Parotitis Epidemica, by Linck, Königsberg; Meningitis Cerebrospinalis Epidemica, by Birkholz, Annaberg; The Toxic Ear, by Beck, Heidelberg; General Pathology, by Fleischmann, Frankfurt; General Symptomatology, by Grünberg, Bonn; Bacteriological Phases, by Streit, Königsberg; Methods of Clinical Examination, by Brühl, Berlin; Photography of the Drum Membrane, by Hegener, Hamburg; Functional Tests of the Acoustic Mechanism, by Schlittler, Basel; Functional Tests of the Static Labyrinth, by Ruttin, Vienna; Roentgen Diagnosis, by Sonnenkalb; Chemnitz; Lumbar Puncture, by Birkholz, Annaberg; General Therapy, by Knick, Leipzig.

This extensive publication should really be called a complete encyclopedia, for it contains most extensive monographs of this long array of subject-matter that are complete in themselves. It is the most ambitious and most complete publication ever undertaken in Otolaryngology and each volume as issued seems to bear testimony of the judgment of both editors and publishers. It incorporates the united strength of the many serious experienced and highly specialized Germanic workers in Otolaryngology in the compass of one glorious effort. M. A. G.

Hay-Fever and Asthma. By Ray M. Balyeat, A. M., M. D., Instructor in Medicine in the University of Oklahoma Medical School; Consulting Physician to St. Anthony Hospital and to the State University Hospital, etc. With 27 illustrations. Philadelphia: F. A. Davis Company, 1926. Price, $2.00.

A timely volume, practically considered, simply presented and containing all the elements of Hay-Fever discussion and technique, including details of desensitization, use of adrenalin, illustrative cases, etc. It is a compendium for both the Hay-Feverite and for the physician interested in its practical disposition. M. A. G.

Local Anesthesia in Otolaryngology and Rhinology. By James Joseph King, A. B., M. D., Assistant Surgeon in Otology, New York Eye and Ear Infirmary; Consulting Otolaryngologist to the French Hospital. Edited by Emil Mayer, M. D. With 21 illustrations and a folded table of cases. New York: Paul B. Hoeber, Inc. Price, $5.00.

This volume has been largely instigated through the untiring efforts and altruism of Dr. Emil Mayer's Committee on Local Anesthesia, functioning for a number of years to determine the consensus of opinion of experienced Otologists in this field.

The technique of local anesthesia in ear, nose and throat surgery has been crystallized; the difference of opinion as to the choice of anesthetics has been analyzed; manner of application and details thereof has been practically presented; the literature has been carefully collected and arranged and the fine work of the Committee of the American Medical Association on the toxic effects of local anesthetics, as offered in their three reports, has been incorporated in this volume. There has also been added the tabulated data of deaths from local anesthetics, as arranged by Dr. Emil Mayer, and the subject-matter has been brought up-to-date in a thoroughly comprehensive manner, and is offered with more detail in technique than has hitherto been published in any textbook.

The drugs considered and fully described are Cocaine, Novocaine, Butyn and Epinephrin, in addition to Morphine and Magnesium Sulphate.

Finally, the author has affectionately dedicated this book to Dr. Emil Mayer, as a token of appreciation of his efforts and service in the collection of this data for the benefit of Otolaryngology. M. A. G.